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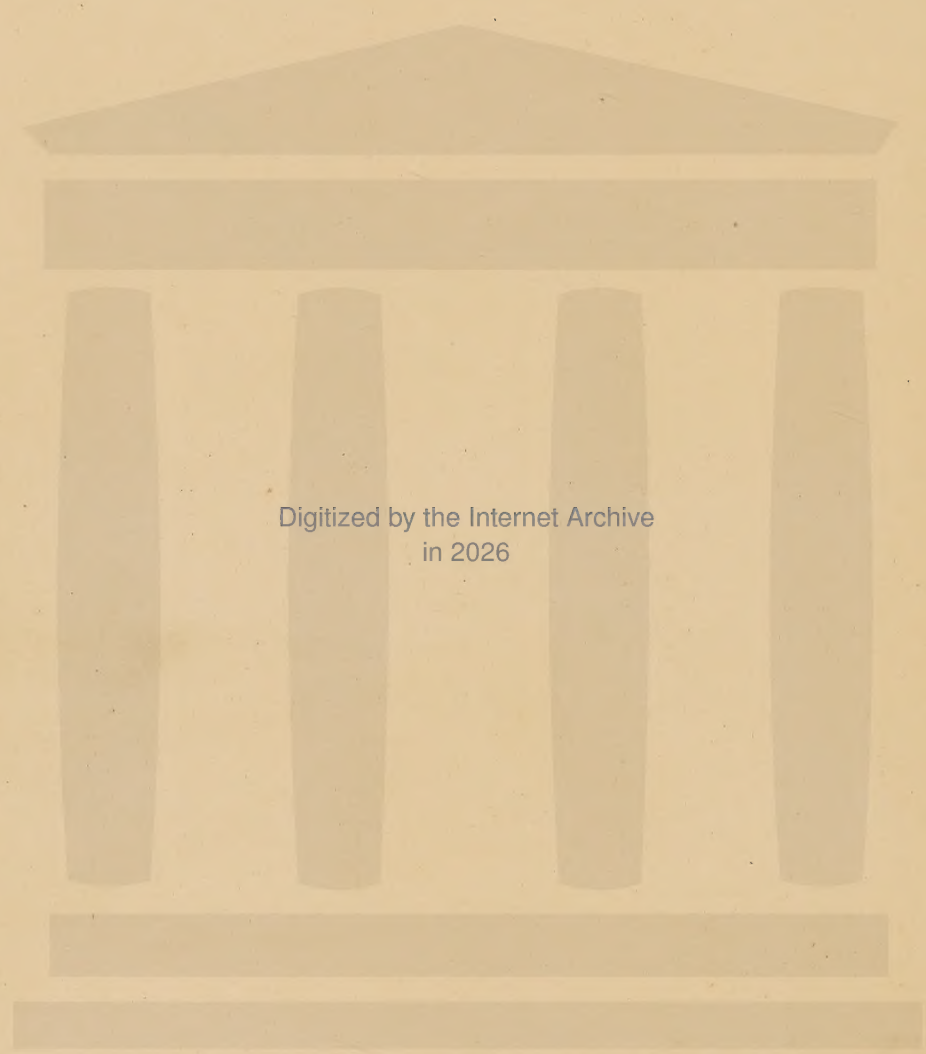

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TRANSACTIONS

OF THE

FIFTY-SEVENTH MEETING

OF THE

AMERICAN SURGICAL ASSOCIATION

HELD AT THE NORTHWESTERN UNIVERSITY
CHICAGO, ILL., MAY 7–8–9, 1936

VOLUME THE FIFTY-FOURTH

EDITED BY

WALTER ESTELL LEE, M.D.

RECORDER OF THE ASSOCIATION

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CONTENTS

Officers of the Association vii

List of Presidents of the Association ix

Fellows of the Association xi

Senior Fellows xxxi

Honorary Fellows xxxiv

Deceased Fellows xxxvi

Address of the President: The Making of a Surgeon. By EUGENE H. POOL, M.D. 1

Report of the Committee to Study Further Problems of Postgraduate Surgical Education in General and the Qualifications for Specialization in General Surgery in Particular. By EVARTS A. GRAHAM, M.D. 12

Studies in Brain Injury: Increased Cerebrospinal Fluid Pressure from Blood in the Cerebrospinal Fluid. By WILLIAM H. PARKER, M.D., and EDWIN P. LEHMAN, M.D. 19

Reactions Following Operations for Hyperthyroidism. By JOHN DEJ. PEMBERTON, M.D. 34

Pericardial Resection in Chronic Constrictive Pericarditis. By EDWARD D. CHURCHILL, M.D. 43

Tumors of the Chest Wall. By KELLOGG SPEED, M.D. 57

Some Advances in the Technic of Thoracoplasty. By JOHN ALEXANDER, M.D. . . 72

The Late Results of Thoracoplasty in the Treatment of Pulmonary Tuberculosis. By B. NOLAND CARTER, M.D. 79

The Use of Intercostal Muscle in the Closure of Bronchial Fistulae. By NORMAN S. SHENSTONE, M.D. 87

Traumatic Surgery of the Lungs and Pleura. By FRANK K. BOLAND, M.D. . . . 99

A Ten Year Study of Empyema in Children. By GROVER C. PENBERTHY, M.D., and CLIFFORD D. BENSON, M.D. 106

Gastric Acidity Following Operations for Gastric and Duodenal Ulcer. By WALTMAN WALTERS, M.D. 112

A Technic for the Management of Gastrojejunal Ulcers With or Without Gastrocolic or Jejunocolic Fistula. By Francis A. C. Scrimger, M.D. 121

Experimental Lymphedema of the Intestinal Tract and Its Relation to Regional Cicatrizing Enteritis. By Frederick Leet Reichert, M.D., and Mary E. Mathes, M.D. 128

The Reduction of the Increasing Mortality and Morbidity in Acute Appendicitis. By Hugh McKenna, M.D. 144

Resection of the Rectum and Rectosigmoid by Single or Graded Procedures. By Fred W. Rankin, M.D. 155

The One Stage Procedure of the Treatment of Carcinoma of the Rectum. By Frederick A. Coller, M.D., and Henry K. Ransom, M.D. 163

Imperforate Anus with Rectovaginal Cloaca. By Harvey B. Stone, M.D. . . . 178

Operative Insulin Crisis in Resection of the Pancreas. By Le Grand Guerry, M.D., and George T. McCutchen, M.D. 189

Benign Cicatricial Strictures of the Bile Ducts. By Ellsworth Eliot, Jr., M.D. 195

Cholecystoduodenostomy Combined with Pyloric Exclusion. By Philip Sandblom, M.S.; George S. Bergh, M.D., and Andrew C. Ivy, M.D. 229

Twenty-five Years' Experience in the Treatment of Peritonitis. By Knut H. Giertz, M.D. 239

The Prevention of Peritoneal Adhesions by Papain. By Alton Ochsner, M.D., and Ambrose Storck, M.D. 263

Delayed Wound Healing Following Nephrectomy for Tuberculosis. By Edwin Beer, M.D., and Leo Edelman, M.D. 275

The Rôle of Surgery in the Treatment of Actinomycosis. By Owen H. Wangensteen, M.D. 279

The Surgical Treatment of Essential Hypertension. By George J. Heuer, M.D. . . 298

The Demonstration of Hormones in Tumors. By Dean Lewis, M.D., and Charles F. Geschickter, M.D. 314

Transurethral Resection. By Herman L. Kretschmer, M.D. 325

Obstructions About the Mesentery in Infants. By John J. Morton, M.D., and T. Banford Jones, M.D. 342

Splenectomy for Hemoclastic Crises. By George M. Curtis, M.D.; Charles A. Doan, M.D., and Bruce K. Wiseman, M.D. 370

The Influence of Heredity in Cancer. By Henry F. Graham, M.D. 383

Acute Perforation of Gastrojejunal Ulcer. By Clarence G. Toland, M.D., and Harold Lincoln Thompson, M.D. 388

Report of Committee on Archives:

Joseph Colt Bloodgood 413

William Bradley Coley 415

John Fox Connors 419

Leonard Freeman 421

Malcolm LaSalle Harris 424

Carl Arthur Hedblom 426

August Frederick Jonas 428

Edward Starr Judd 431

James Tate Mason 434

Francis Stuart Mathews 437

Berkeley George Andrew Moynihan 441

William Johnson Taylor 444

Jonathan Mayhew Wainwright 446

Index 449

NOTICE

This volume of TRANSACTIONS contains the papers read before the Association at the Meeting held in Chicago May 7, 8 and 9, 1936.

The Association assumes no responsibility for the statements and opinions published in this volume.

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319 Longwood Avenue, Boston, Mass.

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180 Fort Washington Avenue, New York City

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1833 Pine Street, Philadelphia, Pa.

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HARVEY B. STONE, M.D.

LIST OF THE PRESIDENTS

OF THE

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ELECTED

1880. ‡SAMUEL DAVID GROSS, M.D., LL.D., D.C.L. (Oxon.). LL.D. (Cantab.), LL.D. (Edin.)—Founder of the Association.
1883. ‡EDWARD MOTT MOORE, M.D., LL.D.
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1897. ‡THEODORE F. PREWITT, M.D.
1898. ‡WILLIAM W. KEEN, A.M., M.D., LL.D., Hon. F.R.C.S. (Eng., Edin. and Ireland), Hon. F.A.C.S.
1899. ‡ROBERT F. WEIR, A.M., M.D., Hon. F.R.C.S. (Eng.)
1900. ‡ROSWELL PARK, M.D.
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1913. WILLIAM J. MAYO, A.M., M.D., D.Sc., F.R.C.S. (Edin., England and Ireland), LL.D. (Tor.).
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1916. ‡SAMUEL J. MIXTER, S.B., M.D.
1917. ‡THOMAS W. HUNTINGTON, A.B., M.D., LL.D.
1918. ‡LEWIS S. PILCHER, A.M., M.D., LL.D.
1919. GEORGE E. BREWER, A.M., M.D., LL.D., Sc.D., F.R.C.S. (Ireland)
1920. ‡JOHN B. ROBERTS, M.D.
1921. JOHN M. T. FINNEY, B.S., M.D.
1922. ‡L. L. McARTHUR, M.D.
1923. GEORGE W. CRILE, A.B., M.D., Ph.D., LL.D.
1924. ‡ALBERT J. OCHSNER, B.S., M.D., F.R.M.S. (Eng. and Ire.), LL.D.
1925. JOHN H. GIBBON, M.D.
1926. HARVEY CUSHING, A.M., M.D., Sc.D., LL.D., F.R.C.S.
1927. EMMET RIXFORD, Sc.B., M.D.
1928. ELLSWORTH ELIOT, Jr., M.D.
1929. FRED B. LUND, M.D.
1930. ALEXANDER PRIMROSE, C.B., M.B., C.M. (Edin.), F.R.C.S. (Eng.), LL.D. (Edin.).
1931. CHARLES H. MAYO, A.M., M.D., LL.D., Sc.D., F.R.C.S. (Eng.), Ch.M.
1932. ARTHUR DEAN BEVAN, M.D.
1933. DANIEL FISKE JONES, A.B., M.D.
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1936. EVARTS A. GRAHAM, A.B., M.D.

FELLOWS

OF THE

AMERICAN SURGICAL ASSOCIATION

May, 1936

* Denotes Original Fellows.
† Denotes Senior Fellows.

ELECTED

1930. Abell, Irvin, A.M., M.D., 321 West Broadway, Louisville, Kentucky. Clinical Professor of Surgery, Medical School, University of Louisville; Surgeon to the Louisville Public Hospital; St. Joseph's Infirmary; Kosair Hospital for Crippled Children; and Children's Free Hospital.

1933. Adson, A. W., Sc.B., M.D., Mayo Clinic, Rochester, Minnesota. Senior Neurosurgeon, Mayo Clinic; Professor of Neurosurgery, Mayo Foundation Graduate School, University of Minnesota.

1935. Alexander, John, B.S., M.A., M.D., F.A.C.S., 2121 Wallingford Road, Ann Arbor, Michigan. Professor of Surgery, University of Michigan; Surgeon-in-Charge, Division of Thoracic Surgery, University of Michigan Hospital; Chief Surgeon, Michigan State Sanatorium.

1929. Allen, Arthur W., A.B., M.D., 264 Beacon Street, Boston, Mass. Instructor in Surgery, Harvard Medical School; Associate Surgeon, Massachusetts General Hospital; Consulting Surgeon to Massachusetts Charitable Eye and Ear Infirmary, Norwood Hospital, Norwood, Mass., and Emerson Hospital, Concord, Mass.

1929. Andrews, Edmund, A.B., M.D., 950 East 59th Street, Chicago, Ill. Professor of Surgery, University of Chicago; Attending Surgeon to Billings Hospital.

1916. Archibald, Edward William, A.B., M.D., C.M., Hon. F.R.C.S. (Eng.), 3106 Westmount Boulevard, Montreal, Canada. Professor of Surgery and Director of the Surgical Department, McGill University; Surgeon, Royal Victoria Hospital; Associate Surgeon, Children's Memorial Hospital. *Vice-President,* 1925, *President,* 1934.

1922. Bailey, Fred Warren, Sc.B., M.D., 1222 Missouri Theatre Building, St. Louis, Mo. Associate Professor of Surgery, St. Louis University School of Medicine; Surgeon to St. John's Hospital; Visiting Surgeon, St. Louis City Hospital; Surgeon and Chief of Staff, Shriners' Hospital.

1920. †Balch, Franklin G., A.M., M.D., 279 Clarendon Street, Boston, Mass. Member of the Board of Consultation, Massachusetts General Hospital; Consulting Surgeon to the Faulkner and Lawrence Memorial Hospitals.

1917. Balfour, Donald Church, M.B., M.D., F.R.A.C.S., LL.D., Mayo Clinic, Rochester, Minn. Professor of Surgery, University of

ELECTED

Minnesota; Chief of Surgery, Mayo Foundation; Surgeon to St. Mary's Hospital, Rochester. *Vice-President,* 1922.

1925. Bancroft, Frederic W., A.B., M.D., 1 East 105th Street, New York, N. Y. Associate Professor of Clinical Surgery, College of Physicians and Surgeons, Columbia University; Surgical Director, Fifth Avenue Hospital; Attending Surgeon, U. S. Veterans' Hospital No. 81; Consulting Surgeon, Lincoln and Harlem Hospitals.

1926. Bazin, Alfred T., C.M., M.D., D.S.O., 4064 Dorchester Street, West, Montreal, Canada. Professor of Surgery, McGill University; Attending Surgeon, Montreal General Hospital; Consulting Surgeon, Champlain Valley Hospital, Plattsburg, N. Y.

1934. Beck, Claude S., A.B., M.D., Lakeside Hospital, Cleveland, Ohio; Associate Professor of Surgery, Western Reserve University School of Medicine; Associate Surgeon, University Hospitals.

1924. Beer, Edwin, A.B., M.D., 45 East Eighty-fifth Street, New York City. Consulting Surgeon, Bellevue Hospital, Fourth Division, and Visiting Surgeon, Mount Sinai Hospital, New York City.

1915. Besley, Frederic A., M.D., 215 North Sheridan Road, Waukegan, Ill. Professor of Surgery, Northwestern University Medical School; Attending Surgeon, Victory Memorial Hospital, Waukegan, and Cook County Hospital, Chicago, Ill.

1900. †Bevan, Arthur Dean, M.D., 122 South Michigan Avenue, Chicago. Professor and Head of the Department of Surgery, Rush Medical College, University of Chicago; Surgeon to Presbyterian Hospital. *President,* 1932.

1931. Beye, Howard Lombard, B.S., M.D., University Hospital, Iowa City, Iowa. Professor and Head of Department of Surgery, College of Medicine, State University of Iowa.

1936. Bigger, Isaac A., M.D., 3310 Loxley Road, Richmond, Va. Professor of Surgery, Medical College of Virginia; Surgeon-in-Chief, Medical College of Virginia Hospital; Surgeon, Crippled Children's Hospital.

1926. Billings, Arthur E., M.D., 2020 Spruce Street, Philadelphia. Clinical Professor of Surgery, Jefferson Medical College; Surgeon to the Bryn Mawr Hospital.

1930. Binney, Horace, A.B., M.D., 370 Commonwealth Avenue, Boston, Mass. Chief Surgeon, Boston City Hospital; Professor of Surgery, Tufts College Medical School, Boston, Mass.

1918. Blair, Vilray Papin, A.B., A.M., M.D., Metropolitan Building, St. Louis, Mo. Professor of Clinical Surgery, Washington University School of Medicine; Professor of Oral Surgery, Washington University School of Dentistry; Associate Surgeon to Barnes, St. Louis, Children's and St. Louis Maternity Hospitals; Attending Specialist in Plastic Surgery, U.S. Veterans' Hospital No. 92.

1906. †Blake, John Bapst, A.M., M.D., 311 Beacon Street, Boston, Mass. Assistant Professor in Surgery, Harvard Medical School; Surgeon-in-Chief to the Boston City Hospital; Consulting Surgeon to Long Island Hospital; Consulting Surgeon to Boston Insane Hospital, and to the Reformatory for Women, Framingham, Mass.

1902. †Blake, Joseph A., A.B., Ph.B., A.M. (Hon.), M.D., R.F.D. No. 1, Franklin, Maine.

1934. Blalock, Alfred, A.B., M.D., Vanderbilt University Hospital, Nashville, Tenn. Associate Professor of Surgery, Vanderbilt Medical School, and Visiting Surgeon to the Vanderbilt University Hospital.

ELECTED

1927. Boland, Frank Kells, A.B., M.D., Sc.D., 478 Peachtree Street, N.E., Atlanta, Georgia. Professor Clinical Surgery, Emory University Medical School; Visiting Surgeon, Emory University Hospital, Grady Municipal Hospital, Crawford W. Long Memorial Hospital; Consulting Surgeon, Georgia Baptist Hospital.

1900. †Brewer, George Emerson, A.M., M.D., LL.D., Sc.D., F.R.C.S. (Ireland), 151 East Seventy-ninth Street, New York City. Emeritus Professor of Surgery in College of Physicians and Surgeons, Columbia University; Consulting Surgeon to the Presbyterian Hospital, Roosevelt Hospital, City Hospital, Woman's Hospital, St. Vincent's Hospital, Knickerbocker Hospital, Fifth Avenue Hospital, Manhattan Eye, Ear, Nose and Throat Hospital, Ophthalmic and Aural Institute, St. Bartholomew's Hospital and Clinic, and House of the Holy Comforter, New York; the Flushing Hospital, Perth Amboy Hospital, the Muhlenberg Hospital of Plainfield, N. J., and Christ Hospital, Jersey City. *Vice-President*, 1913; *President*, 1919.

1917. †Brewster, George W. W., A.B., M.D., 213 Beacon Street, Boston, Mass. Board of Consultation, Massachusetts General Hospital, Boston; Consulting Surgeon, Chelsea Memorial Hospital, Chelsea, Mass.; Leonard Morse Hospital, Natick, Mass.; Milford Hospital, Milford, Mass.; Brockton Hospital, Brockton, Mass.; and Beth Israel Hospital, Boston, Mass.

1915. †Brinsmade, W. B., A.B., M.D., Bedford Hills, N. Y. Consulting Surgeon to St. Christopher's Hospital; Senior Surgeon, Brooklyn Hospital; Commander, M.C., Auxiliary Navy, U.S.A.

1925. Brooks, Barney, Sc.B., M.D., Vanderbilt University Hospital, Nashville, Tenn. Professor of Surgery, Vanderbilt University Medical School; Surgeon-in-Chief, Vanderbilt University Hospital.

1927. Brown, Alfred J., A.B., M.D., Medical Arts Building, Omaha, Nebraska. Professor of Surgery, University of Nebraska College of Medicine; Member of Surgical Staff of Bishop Clarkson Memorial Hospital, Lutheran Hospital, and University of Nebraska Hospital.

1929. Brown, Henry P., Jr., Sc.B., M.D., 1930 Chestnut Street, Philadelphia. Assistant Professor of Surgery, the Graduate School of Medicine, University of Pennsylvania; Surgeon to Children's Hospital and Pennsylvania Hospital; Surgeon to Presbyterian Hospital.

1911. †Bruce, Herbert A., M.D., L.R.C.P. (London), F.R.C.S. (England), 64 Bloor Street East, Toronto, Canada. Professor of Clinical Surgery, University of Toronto; Consulting Surgeon to the Toronto General Hospital; Colonel, Canadian Army Medical Corps and Royal Army Medical Corps; Consulting Surgeon to the British Armies in France, 1917–18.

1929. †Brunn, Harold, M.D., 384 Post Street, San Francisco, Cal. Clinical Professor of Surgery, University of California Medical School; Chief of Surgical Service, San Francisco Hospital and Mt. Zion Hospital; Consulting Surgeon, Southern Pacific Railroad.

1911. †Buchanan, J. J., A.M., M.D., Ph.D., 1409 North Highland Avenue, Pittsburgh, Pa. Professor of Surgery, University of Pittsburgh; Surgeon to Mercy Hospital.

1932. Burdick, Carl G., M.D., 140 East Fifty-fourth Street, New York City. Director Fourth Surgical Division, Bellevue Hospital; Attending Surgeon, Ruptured and Crippled Hospital, New York City; Consulting Surgeon, New York Infirmary for Women and Children, New York City; Clinical Professor of Surgery, New York Uni-

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versity Medical College; Clinical Professor of Oral Surgery, New York University Dental College; Consulting Surgeon, Hospital and Home for Crippled Children, Newark, N. J.

1924. Cabot, Hugh, A.B., M.D., C.M.G., LL.D., Surgeon, Mayo Clinic, Rochester, Minn. Professor of Surgery, Graduate School, University of Minnesota.

1909. †Carrel, Alexis, M.D., Sc.D., Rockefeller Institute for Medical Research, Sixty-sixth Street and York Avenue, New York, N. Y. *Vice-President,* 1919.

1935. Carter, B. Nolan, B.A., M.D., Ph.D., Cincinnati General Hospital, Cincinnati, Ohio. Assistant Director, Surgical Service, Cincinnati General Hospital; Assistant Director, Surgical Service, Children's Hospital; Attending Surgeon, Hamilton County Tuberculosis Sanatorium. Associate Professor of Surgery, University of Cincinnati.

1934. Cave, Henry W., M.D., 107 East Sixty-seventh Street, New York, N. Y. Attending Surgeon, The Roosevelt Hospital; Instructor in Surgery, Columbia University, College of Physicians and Surgeons.

1922. Cheever, David, A.B., M.D., Peter Bent Brigham Hospital, Boston, Mass. Associate Professor of Surgery, Harvard Medical School; Surgeon to the Peter Bent Brigham Hospital.

1932. Churchill, Edward D., B.S., M.A., M.D., Massachusetts General Hospital, Boston, Mass. Chief of West Surgical Service, Massachusetts General Hospital; John Homans Professor of Surgery, Harvard University.

1926. †Clark, Edmund D., M.D., 23 East Ohio Street, Indianapolis, Indiana. University and Bellevue Hospital Medical College, 1891. Professor of Surgery, Indiana University School of Medicine; Consulting Surgeon, Indianapolis City Hospital; Member of Staff University Hospitals—Coleman Hospital for Women, Robert W. Long Hospital and James Whitcomb Riley Hospital for Children; Member of Staff of Methodist Hospital and St. Vincent's Hospital.

1920. Clinton, Marshall, M.D., 135 Linwood Avenue, Buffalo, New York. Associate Professor of Surgery, University of Buffalo, Department of Medicine; Professor of Oral Surgery, University of Buffalo, Dental Department; Attending Surgeon, Buffalo General, Erie County, and Buffalo City Hospitals.

1920. Clopton, Malvern Bryan, M.D., 3720 Washington Boulevard, St. Louis, Mo. Professor of Clinical Surgery, Washington University Medical School; Associate Surgeon, Barnes School; Associate Surgeon, St. Louis Children's Hospital; Chairman of Surgical Staff of St. Luke's Hospital; Consulting Surgeon, Jewish Hospital.

1916. †Codman, Ernest Amory, A.B., M.D., 227 Beacon Street, Boston, Mass.

1928. Coller, Frederick A., Sc.M., M.D., 2105 Wallingford Road, Ann Arbor, Mich. Professor of Surgery, University of Michigan; Director, Department of Surgery, University Hospital.

1936. Colp, Ralph, B.A., M.D., 111 E. 88th St., New York City. Attending Surgeon, Mount Sinai Hospital; Consulting Surgeon, Beekman Street Hospital, New York City.

1930. Copher, Glover H., A.B., M.D., Washington University Medical School, St. Louis, Mo. Associate Professor of Surgery, Washington University; Chief of the Surgical Clinic of the Washington University Dispensary; Visiting Staff of Barnes Hospital; St. Louis Children's; St. Louis Maternity; and St. Louis City Hospital.

1914. †Cotton, Frederick J., A.B., M.D., A.M., 520 Commonwealth Ave-

ELECTED

nue, Boston, Mass. Visiting Surgeon to the Boston City Hospital; Surgeon-in-Chief, Beth Israel Hospital; Associate in Surgery, Harvard Medical School; Consultant in Surgery, United States Public Health Service, Northeastern District; Consultant in Surgery, New England Hospital for Women and Children; Consultant in Surgery, Peterboro Hospital; Eastern Maine General Hospital and Quincy City Hospital.

1905. †Crile, George W., A.B., M.D., Ph.D., LL.D. (Eng.), 2020 East Ninety-third Street, Cleveland, Ohio. Surgeon, Cleveland Clinic Hospital. *President,* 1923.

1935. Curtis, George M., B.A., M.A., Ph.D., M.D., 4690 Sunbury Road, Columbus, Ohio. Professor of Surgery and of Surgical Research, Ohio State University. Attending Surgeon, University Hospital.

1906. †Cushing, Harvey, A.M., Sc.D., M.D., LL.D., F.R.C.S., Yale Medical School, New Haven, Conn. *President,* 1926.

1925. Cutler, Elliott Carr, A.B., M.D., D.S.M. (U.S.A.). Peter Bent Brigham Hospital, 721 Huntington Avenue, Boston, Mass. Moseley Professor of Surgery, Harvard University; Surgeon in Chief, Peter Bent Brigham Hospital.

1925. Dandy, Walter Edward, A.B., A.M., M.D., LL.D. The Johns Hopkins Hospital, Baltimore, Md. Associate Professor of Surgery, Johns Hopkins University, in charge of Neurological Surgery.

1917. Darrach, William, A.B., A.M., M.D., Sc.D., LL.D.. 180 Fort Washington Avenue, New York City. Professor of Clinical Surgery and Dean Emeritus of the Medical Faculty, College of Physicians and Surgeons, Columbia University; Attending Surgeon, Presbyterian Hospital, New York City; Consulting Surgeon, Babies', Beekman Street, Fifth Avenue, and Willard Parker Hospitals, and the Neurological Institute, New York City, the Greenwich Hospital, Greenwich, Conn., and the Morristown Memorial Hospital, Morristown, N. J.

1923. David, Vernon C., A.B., M.D., 59 East Madison Street (Room 1814), Chicago, Ill. Attending Surgeon, Presbyterian and Cook County Hospital; Chairman, Department of Surgery, Rush Medical College and Presbyterian Hospital. *Secretary,* 1935.

1916. Davis, Carl B., A.B., M.D., 122 South Michigan Boulevard, Chicago, Ill. Associate Professor of Clinical Surgery, Rush Medical College; Attending Surgeon, Presbyterian Hospital, Chicago.

1917. †Davis, John Staige, Ph.B., A.M., M.D., Seven Apartments, Baltimore, Md. Associate Professor of Clinical Surgery, Johns Hopkins University; Visiting and Plastic Surgeon, Union Memorial Hospital, Hospital for the Women of Maryland, Children's Hospital; Visiting Surgeon (Plastic Surgeon) and Dispensary Surgeon, Johns Hopkins Hospital.

1919. †Davis, Lincoln, A.B., M.D., 279 Beacon Street, Boston, Mass. *Secretary,* 1926–1931; *Vice-President,* 1932.

1933. Davis, Loyal, M.D., D.Sc., 54 East Erie Street, Chicago, Ill. Professor of Surgery, Chairman of the Division of Surgery, Northwestern University Medical School; Attending Surgeon, Passavant Memorial, Wesley Memorial and Michael Reese Hospitals.

1935. Dinsmore, Robert Scott, B.A., M.D., 2020 East Ninety-third Street, Cleveland, Ohio. Surgeon to the Cleveland Clinic.

1936. Dixon, Claude F., B.S., M.D., M.S., Mayo Clinic, Rochester, Minn. Associate Professor of Surgery, Mayo Foundation, University of Minnesota Medical School Surgeon, Mayo Clinic.

ELECTED

1936. Donovan, Edward J., B.S., M.D., 424 Park Avenue, New York City. Assistant Professor of Surgery, College of Physicians and Surgeons, Columbia University; Attending Surgeon, St. Luke's Hospital, Babies Hospital; Consulting Surgeon to the Northern Westchester Hospital, Mt. Kisco, N. Y., Yonkers General Hospital, Yonkers, N. Y., Fitken Memorial Hospital, Asbury Park, N. J.

1924. Douglas, John, Sc.B., M.D., 568 Park Avenue, New York City. Clinical Professor of Surgery, New York University and Bellevue Medical College; Attending Surgeon, St. Luke's Hospital; Consulting Surgeon to Bellevue, Knickerbocker and Harlem Eye and Ear Hospitals.

1914. †Downes, William A., M.D., 424 Park Avenue, New York City. Professor of Clinical Surgery, Columbia University, College of Physicians and Surgeons; Attending Surgeon, St. Luke's and Babies' Hospitals; Consulting Surgeon, Memorial, Stamford Hospitals, Stamford, Conn., Manhattan State Hospital, Hospital for Ruptured and Crippled, Infirmary for Women and Children, New York, and United Hospital, Portchester, N. Y.

1934. Dragstedt, Lester R., M.D., Ph.D., 5200 Greenwood Avenue, Chicago, Ill. Professor of Surgery, University of Chicago; Attending Surgeon, Albert Merritt Billings Memorial Hospital.

1924. Drennen, Earle, A.B., M.D., 2121 Highland Avenue, Birmingham, Alabama. Attending Surgeon, Hillman Hospital, St. Vincent's Hospital and Children's Hospital.

1919. †Eastman, Joseph Rilus, A.M., Sc.B., M.D., 970 North Meridan Street, Indianapolis, Ind. Professor of Surgery, Indiana University School of Medicine; Visiting Surgeon to City Hospital.

1925. †Eberts, Edmond Melchior, M.D., M.R.C.S. (Eng.), L.R.C.P. (London), 1414 Drummond Street, Montreal, Canada. Professor of Surgery and Clinical Surgery, McGill University; Attending Surgeon, Montreal General Hospital.

1930. Eggers, Carl, M.D., 850 Park Avenue, New York, N. Y. Attending Surgeon, Lenox Hill Hospital, New York Post-Graduate Medical School and Hospital; Consulting Surgeon, Lawrence Hospital, Bronxville, N. Y.; Clinical Professor of Surgery, New York University; Professor of Clinical Surgery, Columbia University.

1926. Eliason, Eldridge Lyon, B.A., M.D., Sc.D., 326 South Nineteenth Street, Philadelphia, Pa. Professor of Clinical Surgery, University of Pennsylvania School of Medicine and University of Pennsylvania Graduate School of Medicine; Surgeon, University of Pennsylvania, Philadelphia General, Presbyterian, Mt. Sinai, and Delaware County Hospitals.

1901. †Eliot, Ellsworth, Jr., M.D., 34 East Sixty-seventh Street, New York, N. Y. Consulting Surgeon to the Grosveneur Hospital, N. Y.; Lawrence Hospital, Bronxville, N. Y.; White Plains Hospital, White Plains, N. Y.; Mountainside Hospital, Montclair, N. J.; Skin and Cancer Hospital, New York City, and Northeastern Dispensary, New York City; Director of Surgery, Knickerbocker Hospital, New York City; *Vice-President*, 1922; *President*, 1928.

1935. Elkin, Daniel Collier, B.A., M.D., 478 Peachtree Street, N. E., Atlanta, Georgia. Professor of Surgery, Emory University. Surgeon-in-Chief, Emory University Hospitals and the Egleston Hospital for Children; Surgeon, Steiner Cancer Clinic.

ELECTED

1936. Eloesser, Leo, B.S., M.D., 490 Post St., San Francisco, Cal. Clinical Professor of Surgery, Medical Department, Stanford University; Surgeon-in-Chief, Stanford University Service, San Francisco Hospital; Consulting Surgeon, Veterans Administration Facility, Fort Miley, San Francisco, U. S. P. H. and Marine Hospital, San Francisco.

1911. †Elting, Arthur W., A.B., M.D., LL.D., 119 Washington Avenue, Albany, N. Y. Professor of Surgery in the Albany Medical College; Surgeon to the Albany Hospital and to the Child's Hospital, Albany, N. Y.; Consulting Surgeon, Horton Memorial Hospital, Middletown, N. Y. *Vice-President,* 1935.

1926. Erdman, Seward, A.B., M.D., 163 East Sixty-fifth Street, New York, N. Y. Assistant Professor of Clinical Surgery, Cornell Medical College; Attending Surgeon, New York Hospital; Consulting Surgeon, New York Infirmary for Women and Children, New York Eye and Ear Infirmary, Memorial Hospital, Morristown, N. J., and Horton Memorial Hospital, Middletown, N. Y.

1896. †Estes, William Lawrence, A.M., M.D., 805 Delaware Avenue, Bethlehem, Pa. Emeritus Surgeon-in-Chief of St. Luke's Hospital.

1927. Estes, William L., Jr., A.B., M.D., Room 401, Wilbur Trust Building, Bethlehem, Pa. Surgeon-in-Chief, St. Luke's Hospital, Bethlehem, Pa.; Urologist to the Baer Hospital, Allentown, Pa.

1898. †Eve, Duncan, A.M., M.D., Eve Building, Nashville, Tenn. Head of the Department and Professor of Surgery and Clinical Surgery, Medical Department, Vanderbilt University; Chief Surgeon of the Louisville & Nashville Railroad; Chief Surgeon of the N. C. and St. Louis R.R.; Attending Surgeon, St. Thomas' Hospital; Consulting Surgeon to Nashville City and Woman's Hospitals.

1899. †Finney, John M. T., M.D., 2947 St. Paul Street, Baltimore, Md. Professor of Surgery, Emeritus, Johns Hopkins University; Visiting Surgeon, Johns Hopkins Hospital, Union Memorial Hospital, Hospital for the Women of Maryland; Brig. General and Chief Consultant, A.E.F.; *President,* 1921.

1932. Finney, John M. T., Jr., B.S., M.D., 2947 St. Paul Street, Baltimore, Md. Associate in Surgery, Johns Hopkins Medical School; Assistant Dispensary Surgeon and Assistant Visiting Surgeon, Johns Hopkins Hospital; Visiting Surgeon, Union Memorial Hospital and Hospital for the Women of Maryland; Consultant in Surgery, Provident Hospital.

1921. †Fisher, William A., M.D., 715 Park Avenue, Baltimore, Md. Associate in Clinical Surgery, Johns Hopkins University; Assistant Visiting Surgeon, Johns Hopkins Hospital; Visiting Surgeon, Union Memorial Hospital, Church Home and Infirmary, Hospital for the Women of Maryland, Children's Hospital School.

1933. Flick, John B., M.D., 1608 Spruce Street, Philadelphia. Surgeon to the Pennsylvania Hospital; Associate Surgeon to Jefferson Hospital; Assistant Surgeon to Bryn Mawr Hospital; Assistant Professor of Surgery, Jefferson Medical College.

1929. Foss, Harold L., M.D., Sc.D. (Hon.), Geisinger Memorial Hospital, Danville, Penna. Surgeon in Chief, Geisinger Memorial Hospital; Surgeon to the Danville State Hospital for the Insane; Surgeon to the Federal Northeastern Penitentiary, Lewisburg, Pa.

1910. †Gage, Homer, A.M., M.D., 8 Chestnut Street, Worcester, Mass. Surgeon-in-Chief to Memorial Hospital; Consulting Surgeon to Worcester City and St. Vincent's Hospitals. *Vice-President,* 1923.

ELECTED

1922. Gallie, William Edward, M.D., F.R.C.S. (Eng.), Medical Arts Building, Toronto, Canada. Professor of Surgery, University of Toronto; Surgeon-in-Chief, Toronto General Hospital; Consulting Surgeon, Hospital for Sick Children, Toronto. *Vice-President,* 1935.

1934. Gatch, Willis D., A.B., M.D., 1933 North Delaware Street, Indianapolis, Ind. Professor of Surgery and Dean, Indiana University School of Medicine; Surgical Staff, St. Vincent's, Methodist Episcopal, Indianapolis City, and Indiana University Hospitals.

1929. Gatewood, Gatewood A. M., M.D., 122 South Michigan Avenue, Chicago, Ill. Associate Clinical Professor of Surgery, Rush Medical College, University of Chicago; Associate Surgeon, Presbyterian Hospital; Attending Surgeon, Cook County Hospital and Highland Park Hospital.

1919. Gaub, Otto Carl, M.D., 1715 Clark Building, Pittsburgh, Pa. Surgeon to Allegheny General Hospital and to Columbia Hospital.

1906. †Gibbon, John H., M.D., 1608 Spruce Street, Philadelphia. Emeritus Professor of Surgery, Jefferson Medical College; Consulting Surgeon, Pennsylvania Hospital and Bryn Mawr Hospital. *Secretary,* 1915–22. *President,* 1925.

1906. †Gibson, C. L., M.D., 1 West Fifty-fourth Street, New York, N. Y. Professor Emeritus of Surgery, Cornell University Medical College; Consulting Surgeon to St. Luke's, City, New York and General Memorial Hospitals; New York State Hospital for Care of Crippled and Deformed Children; South Side Hospital, Bay Shore; Vassar Brothers Hospital, Poughkeepsie. *Vice-President,* 1915.

1928. Goetsch, Emil, Sc.B., M.D., Ph.D., Medical Arts Building, 142 Joralemon Street, Brooklyn, N. Y. Professor of Surgery, Long Island Medical School and Surgeon-in-Chief, Long Island College Hospital, Brooklyn, N. Y.

1934. Grace, Roderick V., A.B., M.D., 100 East Sixty-sixth Street, New York, N. Y. Assistant Professor Clinical Surgery, College of Physicians and Surgeons, Columbia University; Visiting Surgeon, Bellevue Hospital; Senior Associate Surgeon, Hospital for Ruptured and Crippled, New York City.

1920. Graham, Evarts A., A.B., M.D., *President,* 600 South Kingshighway, St. Louis, Mo. Professor of Surgery, Washington University Medical School, St. Louis, Missouri; Surgeon-in-Chief to Barnes Hospital and St. Louis Children's Hospital.

1933. Graham, Henry F., M.D., 474 First Street, Brooklyn, N. Y. Senior Surgeon to the Methodist Episcopal Hospital, Brooklyn, N. Y.

1932. Graham, Roscoe R., M.D., F.R.C.S. (Canada), 1015 Medical Arts Building, St. George Street at Bloor, Toronto 5, Canada. Senior Surgeon, Toronto General Hospital; Assistant Professor of Clinical Surgery, University of Toronto.

1936. Grant, Francis C., A.B., M.D., University Hospital, 34th & Spruce Sts., Philadelphia, Pa. Professor of Neurosurgery, Graduate School of Medicine, University of Pennsylvania.

1911. †Greenough, Robert B., A.B., M.D., 8 Marlborough Street, Boston, Mass. Consulting Surgeon to the Huntingdon Hospital of the Cancer Commission of Harvard University; Member of Board of Consultation, Massachusetts General Hospital. *Secretary,* 1922–26; *Vice-President,* 1927.

1922. Guerry, LeGrand, M.D., D.C.L. (Hon.), LL.D. (Hon.), Sc.D. (Hon.), 1831 Pendleton Street, Columbia, S. C. Surgeon to the Columbia Hospital, Columbia, S. C.

ELECTED

1934. Gurd, Fraser B., B.A., M.D., C.M., 1538 Sherbrooke Street West, Montreal, Que. Associate Surgeon, The Montreal General Hospital; Lecturer in Surgery, McGill University.

1924. Guthrie, Donald, Ph.B., M.D., Sayre, Pa. Surgeon-in-Chief, Robert Packer Hospital.

1916. Haggard, William D., M.D., D.C.L. (Hon.), 340 Doctors Building, Nashville, Tenn. Professor of Clinical Surgery, Vanderbilt University; Surgeon to St. Thomas' Hospital.

1931. Harrington, Stuart Wm., M.D., M.S., Mayo Clinic, Rochester, Minn. Head of a Section on General and Thoracic Surgery at the Mayo Clinic; Associate Professor of Surgery in the Mayo Foundation.

1936. Harris, Robert I., M.C., M.B., F.R.C.S. (Canada), 609 Medical Arts Building, Toronto, Canada. Associate in Surgery, Faculty of Medicine, University of Toronto. Associate Surgeon, Toronto General Hospital; Consulting Surgeon, Toronto Hospital for Consumptives, Freeport Sanatorium, Christie Street Hospital, Department of Pensions and National Health.

1916. †Hartwell, John A., Ph.B., M.D., 27 East Sixty-third Street, New York. Associate Professor of Surgery and Professor of Clinical Surgery, Cornell University Medical College; Former Director of Surgery, Cornell University Division, Bellevue Hospital; Consulting Surgeon to Memorial Hospital, Reconstruction Hospital, New York Infirmary for Women and Children, Lincoln Hospital, Presbyterian Hospital, Lawrence Hospital, Bronxville, N. Y., and United Hospital, Portchester, N. Y.

1926. Harvey, Samuel C., Ph.B., Sc.D. (Hon.), M.D., 789 Howard Avenue, New Haven, Conn. Professor of Surgery on the William H. Carmalt Foundation, Yale University; Surgeon-in-Chief, New Haven Hospital and New Haven Dispensary; Consulting Surgeon, Meriden Hospital, Meriden, Conn.; Consulting Surgeon, Connecticut State Tuberculosis Commission.

1929. Hegner, Casper Frank, M.D., Metropolitan Building, Denver, Col. Assistant Professor of Surgery, University of Colorado School of Medicine; Attending Surgeon, Colorado General Hospital, National Jewish Hospital for Consumptives; Visiting Surgeon, St. Luke's, St. Joseph's, Mercy and Presbyterian Hospitals; Consulting Surgeon, St. Anthony's Hospital and Denver Episcopal Hospital for Children.

1922. Heuer, George J., M.D., 525 East Sixty-eighth Street, New York City. Professor of Surgery, Cornell Medical College; Surgeon-in-Chief, New York Hospital.

1922. Hitzrot, James Morley, A.B., M.D., 126 East Thirty-seventh Street, New York City. Consulting Surgeon, New York Hospital, Hospital for Ruptured and Crippled, the Community Hospital, Nassau Hospital, Mineola; North Shore Community, Glen Cove, L. I.; Director of Surgery, Manhattan General Hospital.

1918. †Hodge, Edward Blanchard, A.B., M.D., 2019 Spruce Street, Philadelphia, Pa. Surgeon to the Presbyterian and Germantown Hospitals; Associate Surgeon, Pennsylvania Hospital; Surgeon-in-Chief, Chester County Hospital; Associate Surgeon, Widener Memorial School.

1923. Homans, John, A.M., M.D., Peter Bent Brigham Hospital, 721 Huntington Avenue, Boston, Mass. Assistant Professor of Sur-

ELECTED

gery, Harvard Medical School; Surgeon to the Peter Bent Brigham Hospital.

1936. Horrax, Gilbert, B.A., M.D., Sc.D., 605 Commonwealth Ave., Boston, Mass. Instructor in Neurology, Harvard Medical School for Graduates. Neurological Surgeon to New England Deaconess and New England Baptist Hospitals, Boston; Chief of Neurological Service of Lahey Clinic, Boston.

1924. Horsley, J. Shelton, M.D., LL.D., 617 West Grace Street, Richmond, Va. Attending Surgeon, St. Elizabeth's Hospital.

1909. †Hutchinson, James P., M.D., Media, Pa.

1930. Jennings, John Edward, M.D., 23 South Portland Avenue, Brooklyn, N. Y. Senior Surgeon, Brooklyn Hospital; Surgeon, St. Peter's Hospital; Surgeon-in-Chief, Cumberland Hospital; Consulting Surgeon, St. John's and Mary Immaculate Hospitals.

1917. †Jones, Daniel Fiske, A.B., M.D., 195 Beacon Street, Boston, Mass. Associate in Surgery, Harvard Medical School; Consulting Surgeon, Massachusetts General Hospital and New England Deaconess Hospital; Surgeon-in-Chief, Palmer Memorial Hospital; Consulting Surgeon, New England Hospital for Women and Children and Beth Israel Hospital; Boston City Hospital, Union Hospital, Fall River Hospital, Leo Minister Hospital, Sturdy Hospital, Attleboro Hospital, Addison Gilbert Hospital, Gloucester, Noble Hospital, Westfield. *Vice-President,* 1930; *President,* 1933.

1910. †Jopson, John Howard, M.D., Waverly Mills, S. C. *Recorder,* 1922 to 1930.

1930. Joyce, Thomas M., M.D., 2823 N. W. Cumberland Road, Portland, Oregon. Professor of Surgery and Head of the Department of Surgery, University of Oregon Medical School; Chief Surgeon to the Multnomah County Hospital and Doernbecher Memorial Hospital for Children; Surgeon to St. Vincent's Hospital and the Portland Clinic.

1913. Kanavel, Allen B., A.B., M.D., Sc.D. (Hon.), 54 East Erie Street, Chicago, Ill. Professor of Surgery, Northwestern University Medical School; Attending Surgeon, Wesley Memorial and Passavant Hospitals.

1923. Keller, William L., M.D., Walter Reed General Hospital, Washington, D. C. Colonel, M.C., U.S.A.; Professor of Military Surgery, Army Medical School, Army Medical Center, Washington, D. C.; Chief of the Surgical Service, Walter Reed General Hospital.

1935. Kennedy, Robert H., B.A., M.D., 115 East Sixty-first Street, New York City. Associate Professor of Clinical Surgery, New York Post-Graduate Medical School, Columbia University; Surgical Director, Beekman Street Hospital; Associate Surgeon, New York Post-Graduate Hospital.

1924. Kerr, Harry Hyland, M.D., C.M., 1744 N Street, N. W., Washington, D. C. Clinical Professor of Surgery, the George Washington University School of Medicine; Attending Surgeon to the Garfield Memorial and Children's Hospitals; Consulting Surgeon to Providence, Columbia Hospital for Women, Episcopal Eye, Ear, Nose and Throat, and U. S. Veterans' Bureau Hospitals; Lieut.-Colonel Medical Reserve Corps, U.S.A.

1935. Key, J. Albert, B.A., M.D., Washington University School of Medicine, Department of Surgery, St. Louis, Mo. Clinical Professor of Orthopedic Surgery, Washington University; Visiting Orthopedic

ELECTED

Surgeon to the Barnes, Jewish, St. Louis Children's and St. Louis City Hospitals, and the Shriners' Hospital for Crippled Children.

1930. Koch, Sumner L., A.M., M.D., 54 East Erie Street, Chicago, Ill. Associate Professor of Surgery, Northwestern University Medical School; Attending Surgeon, Passavant Memorial and Cook County Hospitals.

1931. Ladd, William E., A.B., M.D., 300 Longwood Avenue, Boston, Mass. Chief of Surgical Service, Children's Hospital, Boston; Clinical Professor of Surgery, Harvard Medical School.

1925. Lahey, Frank H., M.D., 605 Commonwealth Avenue, Boston, Mass. Surgeon, N. E. Deaconess' Hospital and N. E. Baptist Hospital.

1923. Lee, Walter Estell, M.D., *Recorder,* 1833 Pine Street, Philadelphia, Pa. Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; Surgeon to the Pennsylvania, Germantown, Bryn Mawr, Children's, Graduate, and Burlington County Hospitals; Lieut.-Colonel, Medical Reserve Corps, U.S.A.

1931. Lehman, Edwin Partridge, A.B., M.D., University of Virginia Hospital, University, Va. Professor of Surgery and Gynecology, University of Virginia Department of Medicine; Chief Surgeon University of Virginia Hospital.

1935. Leland, George A., Jr., B.A., M.D., 144 Commonwealth Avenue, Boston, Mass. Associate in Surgery, Medical School of Harvard University; Visiting Surgeon, Massachusetts General Hospital, Collis P. Huntingdon Memorial Hospital and Palmer Memorial Hospital; Consulting Surgeon, Massachusetts Charitable Eye and Ear Infirmary, Addison Gilbert Hospital, Gloucester.

1933. Lenhart, Carl H., Ph.B., M.D., 2065 Adelbert Road, Cleveland, Ohio. Professor of Surgery, Western Reserve University; Director of Surgery, University Hospitals (At Lakeside Hospital); Director of the Division of Surgery of Cleveland City Hospital.

1912. Lewis, Dean D., M.D., Johns Hopkins Hospital, Baltimore, Md. Professor of Surgery, Johns Hopkins University. *Vice-President,* 1928.

1910. †Lilienthal, Howard, A.B., M.D., 52 East Eighty-second Street, New York. Consulting Surgeon to Mount Sinai Hospital, Bellevue Hospital, Hospital for Joint Disease, New York Infirmary for Women and Children, and Jersey City Medical Centers; Formerly Professor of Clinical Surgery, Cornell University Medical College; Formerly Lieut.-Colonel, M.C., U.S.A., A.E.F.

1916. Lower, William E., M.D., 2020 East Ninety-third Street, Cleveland, Ohio. Cleveland Clinic Hospital; Visiting Surgeon, Lutheran Hospital.

1935. Lund, Charles C., B.A., M.D., 319 Longwood Avenue, Boston, Mass. Assistant Professor of Surgery, Harvard Medical School; Assistant Visiting Surgeon, Boston City Hospital; Surgeon, Collis P. Huntingdon Memorial Hospital.

1910. †Lund, Fred Bates, M.D., 319 Longwood Avenue, Boston, Mass. Consulting Surgeon, Boston City Hospital; Consulting Surgeon, Burbank Hospital, Fitchburg; Newton Hospital, Newton; Surgeon-in-Chief, Carney Hospital, Boston; late Lieut.-Colonel, M.C., U.S.A.; Lieut.-Colonel, Medical Reserve Corps, U.S.A. *President,* 1930.

1917. Lyle, Henry H. M., M.D., M.S., 33 East Sixty-eighth Street, New York City. Professor of Clinical Surgery, Cornell Medical University; Attending Surgeon, St. Luke's Hospital; Director and

ELECTED

Chief Surgeon, Cancer Division, Stuyvesant Square Hospital (Formerly New York Skin & Cancer Hospital); Consulting Surgeon, U. S. Veterans' Hospital, Dist. No. 2, Elizabeth A. Horton Memorial Hospital, Middletown, N. Y., State Reconstruction Hospital, Cornwall Hospital (Cornwall, N. Y.), Newburgh Hospital, N. Y., First Army, U.S.A.; Associate Surgeon, New York Hospital.

1934. LYNN, FRANK S., M.D., 101 West Read Street, Baltimore, Md. Professor of Clinical Surgery University of Maryland; Visiting Surgeon, University Hospital, Bon Secours Hospital and Baltimore City Hospital.

1932. MCCLURE, ROY D., B.A., M.D., 1490 Iroquois Avenue, Detroit, Mich. Surgeon-in-Chief, Henry Ford Hospital; Consulting Surgeon, Receiving Hospital.

1930. MCGLANAN, ALEXIUS, 115 West Franklin Street, Baltimore, Md., A.M., M.D., LL.D., (Hon.). Consulting Surgeon, Mercy Hospital, Baltimore, Md., Professor of Surgery, University of Maryland.

1917. †MCGUIRE, STUART, M.D., LL.D., 1000 West Grace Street, Richmond, Va. Emeritus Professor of Surgery of Medical College of Virginia; Surgeon to St. Luke's Hospital; Consulting Surgeon to Memorial Hospital. *Vice-President*, 1927.

1925. MCKENNA, HUGH, Sc.B., M.D., 700 North Michigan Avenue, Chicago, Ill. Assistant Professor of Surgery, Northwestern University Medical School; Executive Attending Surgeon, St. Joseph's Hospital.

1932. MCKITTRICK, LELAND S., B.S., M.D., 205 Beacon Street, Boston, Mass. Surgeon, Palmer Memorial Hospital and New England Deaconess Hospital; Assistant Surgeon, Massachusetts General Hospital; Surgeon-in-Chief, House of the Good Samaritan; Consulting Surgeon, Sturdy Memorial Hospital, Attleboro, Mass.; Instructor, Harvard Graduate School.

1935. MACFEE, WILLIAM FRANK, B.A., M.D., 1215 Fifth Avenue, New York City. Associate Professor of Clinical Surgery, Columbia University; Associate Attending Surgeon, St. Luke's Hospital and New York Post-Graduate Hospital.

1914. †MACLAREN, MURRAY, A.B., M.B., C.M., M.D., M.R.C.S. (Eng.), C.M.G., C.O.A., LL.D., M.P., 75 Coburg Street, St. John's, N. B., Canada. Surgeon to the General Public Hospital; Colonel, C.A.M.C. Minister of Pensions and National Health, Ottawa.

1920. MAES, URBAN, 1671 Octavia Street, New Orleans, La. Professor of Surgery and Director of the Department, Louisiana State University Medical Center; Senior Visiting Surgeon, Charity Hospital; Senior Associate in Surgery, Touro Infirmary.

1898. †MARTIN, EDWARD, A.M., M.D., LL.D., Sc.D., 135 South Eighteenth Street, Philadelphia, Pa. Professor of Surgical Physiology, University of Pennsylvania; Consulting Surgeon to the State Hospitals, Norristown and Wernersville, Pa.; Colonel, Auxiliary Army, U.S.A.

1915. †MARTIN, WALTON, PH.B., M.D., 230 East Forty-ninth Street, New York. Professor of Clinical Surgery, Columbia University; Consulting Surgeon, St. Luke's Hospital, Home for Incurables, White Plains Hospital, Loomis Sanatorium and Vassar Brothers' Hospital, Poughkeepsie.

1931. MASON, JAMES MONROE, M.D., Sc.D., *Vice-President*, 2721 Niazuma Ave., Birmingham, Alabama. Surgeon-in-Chief, Hillman Hospital; Attending Surgeon, Children's Hospital; Attending Abdominal Sur-

ELECTED

geon, St. Vincent's Hospital; Senior Consultant in Surgery, Employees Hospital, Fairfield, Ala.

1934. Masson, James Carruthers, M.B., M.D., 724 Fourth Street, S.W., Rochester, Minn.; Head of a Section of Surgery Mayo Clinic; Associate Professor of Surgery, The Mayo Foundation, University of Minnesota; Surgeon St. Mary's and Colonial Hospitals.

1895. †Matas, Rudolph, M.D., LL.D., Sc.D., F.R.C.S. (Hon., Eng.), 2255 St. Charles Avenue, New Orleans, La. Professor of Surgery (Emeritus) 1928, Tulane University of Louisiana, School of Medicine; Professor (Emeritus) Graduate Medical School, Tulane University; Chief, Surgical Division, Touro Infirmary; Consulting Surgeon, Charity Hospital; Consulting Surgeon, Eye, Ear, Nose and Throat Hospital; Major, Medical Reserve Corps, U.S.A. *Vice-President,* 1901; *President,* 1909.

1903. Mayo, Charles H., A.M., M.D., LL.D. (University of Maryland, Kenyon College, Northwestern University, University of Edinburgh, Queen's University, Belfast, University of Manchester, Hamline University), Sc.D. (Princeton University, University of Pennsylvania, University of Leeds), Ch.M. (Trinity College, Dublin), Ph.D. (Detroit College of Medicine and Surgery), M.D. (University of Havana), F.R.C.S. (Eng. and Ireland). Professor of Surgery, Medical School and Graduate School (Mayo Foundation), University of Minnesota; Surgeon and Associate Chief of Staff, Mayo Clinic, Mayowood, Rochester, Minnesota. *President,* 1931.

1899. †Mayo, William J., A.M., M.D. (Michigan); M.D. in Surgery, Trinity College, Dublin; M.D. in Surgery, University of Havana, Cuba; Sc.D. (Columbia, Michigan, Harvard, Northwestern, Marquette, Leeds, England); LL.D., F.R.C.S. (England, Edinburgh, and Ireland); LL.D. (Toronto, Can., McGill, Can., University of Aberdeen, Scotland, Pennsylvania, Maryland, Pittsburgh, Temple, Carleton, Manchester, England); F.R.C.S. (England, Surg. Gr. Br. and Ire.; A.M.A., F.A.C.S., 701 Fourth Street, S. W., Rochester, Minnesota. Surgeon and Chief of Staff to Mayo Clinic. *Vice-President,* 1903; *President,* 1913.

1934. Meigs, Joe Vincent, A.B., M.D., 264 Beacon Street, Boston, Mass. Instructor in Surgery, Harvard Medical School; Assistant Visiting Surgeon, Massachusetts General Hospital; Associate Surgeon, Collis P. Huntington Memorial Hospital; Surgeon, Pondville Hospital (Massachusetts State Cancer Hospital), Walpole, Mass.; Assistant Surgeon, Palmer Memorial Hospital, Boston, Mass.

1932. Miller, Edwin M., M.D., 700 North Michigan Avenue, Chicago, Ill. Consulting Surgeon, Durand Memorial Hospital; Attending Surgeon, Cook County Hospital; Assistant Attending Surgeon, Presbyterian Hospital; Associate Clinical Professor of Surgery, Rush Medical College.

1928. Miller, Richard H., A.B., M.D., 264 Beacon Street, Boston, Mass. Assistant Professor of Surgery, Harvard Medical School; Visiting Surgeon, Massachusetts General Hospital; Consulting Surgeon, Massachusetts Eye and Ear Infirmary, Boston Lying-in Hospital, Heywood Hospital, and Leominster Hospital.

1919. Miller, Robert Talbott, A.B., M.D., 9 East Chase Street, Baltimore, Md. Associate Professor of Surgery, Johns Hopkins University; Associate Surgeon, Johns Hopkins Hospital.

1917. †Mitchell, Charles F., M.D., 2003 Pine Street, Philadelphia, Pa. Surgeon-in-Chief, Pennsylvania Hospital; Attending Surgeon to

ELECTED

the Germantown and the Bryn Mawr Hospitals; Consulting Surgeon, Chestnut Hill Hospital.

1912. †Mitchell, James F., A.B., M.D., 1344 Nineteenth Street, Washington, D. C. Clinical Professor of Surgery, Georgetown and George Washington Universities; Chief Surgeon, Emergency Hospital. *Vice-President*, 1920.

1924. Mixter, Charles G., *Secretary*, Sc.B., M.D., 319 Longwood Avenue, Boston, Mass. Assistant Professor of Surgery, Harvard Medical School; Surgeon-in-Chief, Beth Israel Hospital; Visiting Surgeon, Children's Hospital; Consulting Surgeon, Leonard Morse Hospital, Natick; Jordan Hospital, Plymouth; Benjamin Stickney Cable Hospital, Ipswich; Cape Cod Hospital, Hyannis; New Hampshire Memorial Hospital for Women and Children, Concord, New Hampshire; Morton Hospital, Taunton.

1920. Mixter, William Jason, Sc.B., M.D., 319 Longwood Avenue, Boston, Mass. Instructor in Surgery, Courses for Graduates, Harvard Medical School; Visiting Surgeon, Massachusetts General Hospital; Consulting Surgeon, Massachusetts Eye and Ear Infirmary; Neurological Surgeon, Beth Israel Hospital; Consulting Surgeon, Leonard Morse Hospital, Natick; Benjamin Stickney Cable Hospital, Ipswich; Cape Cod Hospital, Hyannis.

1934. Montgomery, Albert H., A.B., M.D., 122 South Michigan Avenue, Chicago, Ill. Associate Clinical Professor of Surgery, Rush Medical College of the University of Chicago; Attending Surgeon and Chief of Surgical Service, Children's Memorial Hospital; Associate Attending Surgeon, Presbyterian Hospital; Attending Surgeon, Cook County Hospital.

1925. Moore, Edward Clarence, M.D., Moore-White Clinic, 511 South Bonnie Brae Street, Los Angeles, Calif. Consulting Surgeon, Children's Hospital and Los Angeles General Hospital; Surgeon to Hollywood Hospital, Hospital of the Good Samaritan, California Lutheran Hospital; Vice-Chairman, California Lutheran Hospital.

1927. Morton, John Jamieson, Jr., A.B., M.D., 18 Audubon Street, Rochester, N. Y. Professor of Surgery, School of Medicine and Dentistry, University of Rochester; Surgeon-in-Chief, Strong Memorial and Municipal Hospitals, Rochester, N. Y.

1919. Muller, George P., M.D., 1930 Spruce Street, Philadelphia, Pa. Professor of Clinical Surgery, Graduate School of Medicine, University of Pennsylvania; Surgeon to Misericordia and Lankenau Hospitals.

1913. †Murphy, Fred T., A.B., A.M., M.D., 4350 Penobscot Building, Detroit, Michigan.

1928. Naffziger, Howard C., Sc.M., M.D., 384 Post Street, San Francisco, Calif. Clinical Professor of Surgery, University of California Medical School; Neurological Surgeon to the University Hospital; Consulting Neurological Surgeon to the San Francisco Hospital, the Children's Hospital, the U. S. Public Health Hospital, the Letterman General Hospital, and the U. S. Veterans' Bureau Hospital, Palo Alto, Calif.

1903. †Neilson, Thomas R., M.D., 1937 Chestnut Street, Philadelphia, Pa. Surgeon Emeritus to the Episcopal Hospital; Consulting Surgeon to St. Christopher's Hospital for Children; Emeritus Professor of Genito-urinary Surgery in the University of Pennsylvania.

1934. Newton, Francis C., A.B., M.D., 319 Longwood Avenue, Boston,

ELECTED

Mass. Instructor in Surgery, Harvard Medical School; Senior Associate Surgeon, Peter Bent Brigham Hospital.

1931. Ochsner, Alton, A.B., M.D., Department of Surgery, Tulane University School of Medicine, 1430 Tulane Avenue, New Orleans, La. Professor of Surgery, Tulane University School of Medicine; Visiting Surgeon, Charity Hospital and Touro Infirmary; Consulting Surgeon, Illinois Central Hospital, New Orleans.

1900. †Oliver, J. C., M.D., 628 Elm Street, Cincinnati, Ohio. Emeritus Professor of Surgery, Medical College, University of Cincinnati; Surgeon to the Christ Hospital.

1915. †Olmsted, Ingersoll, M.D., 215 James Street, South, Hamilton, Ontario. Surgeon to City Hospital.

1929. Orr, Thomas Grover, A.B., M.D., University of Kansas Hospital, Thirty-ninth and Hudson Road, Kansas City, Kansas. Professor of Surgery, University of Kansas School of Medicine; Surgeon-in-Chief, University of Kansas, Bell Memorial Hospital.

1936. Oughterson, Ashley W., M.S., M.D., 789 Howard Ave., New Haven, Conn. Associate Professor of Surgery, Yale University School of Medicine; Associate Surgeon, New Haven Hospital and New Haven Dispensary.

1923. Palmer, Dudley White, ScB., M.D., 707 Race Street Building, Cincinnati, Ohio. Professor of Clinical Surgery, University of Cincinnati, Medical Department; Attending Surgeon, The Christ Hospital, The Good Samaritan Hospital, and The Cincinnati General Hospital. Associate Surgeon, The Children's Hospital.

1935. Parsons, William Barclay, B.A., M.D., 208 East Seventy-second Street, New York City. Assistant Professor of Surgery, Columbia University, College of Physicians and Surgeons; Associate Attending Surgeon, Presbyterian Hospital.

1933. Payne, Robert Lee, M.D., Medical Arts Building, Norfolk, Virginia. Senior Surgeon Norfolk Protestant Hospital and St. Vincent's Hospital.

1929. Pemberton, John de Jarnette, A.B., Sc.M., M.D., LL.D., Rochester, Minn. Associate Professor of Surgery, Mayo Foundation, University of Minnesota; Surgeon to Mayo Clinic.

1934. Penberthy, Grover C., M.D., 1551 Woodward Avenue, Detroit, Michigan. Associate Professor of Surgery, Medical School, Wayne University; Director of General Surgery Children's Hospital of Michigan; Surgeon, Harper and Herman Kiefer Hospitals; Surgeon Michigan Mutual Hospital; Consulting Surgeon, Receiving Hospital.

1936. Penfield, Wilder, M.D., M.A., B.Sc., D.Sc. (Oxon), F.R.C.S. (Canada), Montreal Neurological Institute, Montreal Canada. Professor of Neurology and Neurosurgery, McGill University; Director, Montreal Neurological Institute; Neurological Surgeon to Royal Victoria and Montreal General Hospitals.

1919. Percy, Nelson Mortimer, M.D., 2051 Sedgwick Street, Chicago, Ill. Professor of Clinical Surgery, University of Illinois, College of Medicine; Surgeon-in-Chief to the Augustana Hospital; Senior Attending Surgeon to St. Mary's of Nazareth Hospital.

1925. Pfeiffer, Damon B., A.B., M.D., 1822 Pine Street, Philadelphia, Pa. Lecturer in Surgery, University of Pennsylvania Medical School; Assistant Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; Surgeon, Lankenau and Abington

ELECTED

Memorial Hospitals; Consulting Surgeon, Woman's Hospital of Philadelphia.

1917. PHEMISTER, DALLAS B., Sc.B., M.D., 950 East Fifty-ninth Street, Chicago, Ill. Professor and Chairman of the Department of Surgery, University of Chicago; and Surgeon-in-Chief, Billings Hospital. *Vice-President,* 1930.

1931. PICKHARDT, OTTO C., A.B., M.D., 117 East Eightieth Street, New York, N. Y. Clinical Professor of Surgery, New York University; Surgeon to the Lenox Hill Hospital; Consulting Surgeon, Lutheran Hospital and Isabella Home.

1914. †PLUMMER, SAMUEL C., A.B., M.D., 5844 Stony Island Avenue, Chicago, Ill. Attending Surgeon, St. Luke's Hospital; Chief Surgeon, C. R. I. & P. Railway Co.

1914. POOL, EUGENE H., A.B., M.D., 107 East Sixtieth Street, New York City. Clinical Professor of Surgery, Columbia University College of Physicians and Surgeons; Professor of Clinical Surgery, Cornell University Medical College; Senior Attending Surgeon, New York Hospital; Consulting Surgeon, French, Woman's, Presbyterian, Harlem and New York Orthopedic Hospitals, Home for Incurables, New York Eye and Ear Infirmary, New York Infirmary for Women and Children, Berwind Free Outdoor Maternity Clinic, New York State Hospital Central Islip, United Hospital, Portchester, N. Y., North County Community Hospital, Glen Cove, N. Y., Elizabeth A. Horton Memorial Hospital, Middletown, N. Y., Monmouth Memorial Hospital, Long Branch, N. J.; Surgeon-in-Chief Emeritus, Ruptured and Crippled Hospital. *Treasurer,* 1924–1933; *President,* 1935.

1908. †PRIMROSE, ALEXANDER, C.B., M.B., C.M. (Edin.), F.R.C.S. (Eng.), LL.D. (Edin.), 518 Medical Arts Building, Bloor and St. George Streets, Toronto, Canada. Consulting Surgeon to the Toronto General Hospital and Consulting Surgeon to the Hospital for Sick Children, Toronto; Dean of the Faculty of Medicine (1920–1932) and Emeritus Professor of Clinical Surgery in the University of Toronto. *Vice-President,* 1922–1926; *President,* 1930.

1928. RANKIN, FRED W., A.M., M.D., 410 Security Trust Building, Lexington, Ky.

1935. REICHERT, FREDERICK LEET, PH.B., M.D., Stanford University Hospital, Clay and Webster Streets, San Francisco, California. Professor of Surgery, Stanford University School of Medicine.

1926. REID, MONT ROGERS, A.B., M.D., 1908 Dexter Avenue, East Walnut Hills, Cincinnati, Ohio. Associate Professor of Surgery, University of Cincinnati College of Medicine; Assistant Director, Surgical Service, Cincinnati General Hospital, Children's Hospital and Consulting Surgeon to the U. S. Diagnostic Centre, Cincinnati, Ohio.

1923. †RICHARDSON, EDWARD P., A.B., M.D., Massachusetts General Hospital, Boston, Mass. Formerly John Homans Professor of Surgery, Harvard Medical School; Chief of West Surgical Service, Massachusetts General Hospital.

1931. RIENHOFF, WILLIAM FRANCIS, JR., A.B., M.D., 1201 North Calvert Street, Baltimore, Md. Associate in Surgery, Johns Hopkins University; Instructor in Anatomy, Johns Hopkins Medical School; Visiting Surgeon Johns Hopkins Hospital.

1924. RITCHIE, HARRY P., PH.B., M.D., 914 Lowry Building, St. Paul, Minn. Associate Professor of Surgery, University of Minnesota Medical School; Attending Surgeon, University Hospital, Minne-

ELECTED

apolis; Miller Staff of St. Luke's and Miller Hospitals, and of Children's Hospital, St. Paul.

1901. †Rixford, Emmet, Sc.B., M.D., 1795 California Street, San Francisco, Calif. Professor of Surgery in Stanford University School of Medicine; Professor Emeritus since 1930; Consulting Surgeon to San Francisco and Lane Hospitals; Member of the Society of Clinical Surgery. *Vice-President,* 1905; *President,* 1927.

1929. Robertson, D. E., M.D., *Vice-President,* 1012 Medical Arts Building, St. George Street at Bloor, Toronto, 5, Ontario. Surgeon-in-Chief to Hospital for Sick Children; Associate in Surgery, University of Toronto; Consultant in Orthopædic Surgery, Department of Pensions and National Health.

1924. Rodman, J. Stewart, M.D., 1310 Medical Arts Building, Philadelphia. Professor of Surgery, Woman's Medical College of Pennsylvania; Surgeon-in-Chief, Woman's Medical College Hospital; Attending Surgeon, Bryn Mawr Hospital; Associate Surgeon, Presbyterian Hospital; Major, Auxiliary Army, U.S.A.

1917. †Rowan, Charles J., A.B., M.D., 501 Eighteenth Street, Santa Monica, Calif. Professor and Head of Department of Surgery, College of Medicine, State University of Iowa.

1922. Royster, Hubert A., A.B., M.D., Sc.D. (Hon.), 127 S. Hargett Street, Raleigh, N. C. Surgeon to Rex Hospital; Surgeon-in-Chief to St. Agnes' Hospital; Consulting Surgeon to Dix Hill State Hospital.

1936. Schmidt, Erwin R., B.A., M.D., 1937 Arlington Place, Madison, Wisconsin. Professor of Surgery and Head of Division of Surgery, University of Wisconsin Medical School; Chief Surgeon, State of Wisconsin General Hospital, Madison, Wisconsin.

1928. Schwyzer, Arnold, M.D., 127 West Seventh Street, St. Paul, Minn. Professorial Lecturer emeritus, University of Minnesota; Surgeon, St. Joseph's Hospital, St. Paul, Minn.

1933. Scott, W. J. Merle, A.B., A.M., M.D., 260 Crittenden Boulevard, Rochester, N. Y. Associate Professor of Surgery, University of Rochester; Associate Surgeon, Strong Memorial and Rochester Municipal Hospitals.

1930. Scrimger, Francis, A.C., V.C., A.B., M.D., C.M., 814 Medical Arts Building, 85 Redpath Crescent, Montreal, P.Q., Canada. Lecturer in Surgery, McGill University; Assistant Surgeon, Royal Victoria Hospital; Surgeon, Children's Memorial Hospital, Montreal.

1909. †Scudder, Charles L., M.D., A.B., Ph.B., 374 Commonwealth Avenue, Boston, Mass. Consulting Surgeon, Massachusetts General Hospital; former Assistant Professor of Surgery, Harvard University Medical School.

1919. †Seelig, M. G., A.B., M.D., 4952 Park View Place, St. Louis, Mo. Professor of Clinical Surgery, Washington University School of Medicine; Assistant Surgeon, Barnes Hospital; Visiting Surgeon to the Jewish Hospital.

1934. Shenstone, Norman Straham, B.A., M.D., 904 Medical Arts Building, Toronto, Canada. Assistant Professor of Clinical Surgery, University of Toronto; Senior Attending Surgeon Toronto General and St. John's Hospitals.

1923. Shipley, Arthur M., Sc.D., M.D., 507 Edgevale Road, Roland Park, Baltimore, Md. Professor of Surgery and Head of the Department of Surgery, University of Maryland; Chief Surgeon, University Hospital, City Hospitals and Sydenham Hospital; Visiting Sur-

ELECTED

geon, Union Memorial Hospital, Hospital for Women of Maryland, Mercy Hospital and Maryland General Hospital.

1926. Simmons, Channing C., M.D., 205 Beacon Street, Boston, Mass. Associate in Surgery, Harvard Medical School; Visiting Surgeon, Massachusetts General Hospital, Surgeon-in-Chief, Collis P. Huntington Memorial Hospital.

1935. Singleton, Albert O., B.S., M.D., F.A.C.S., 1602 Avenue J, Galveston, Texas. Professor of Surgery, Medical Dept., University of Texas; Chief Surgeon, John Sealy Hospital.

1931. Smith, Morris K., A.B., M.D., 33 East Sixty-eighth Street, New York City. Assistant Professor Clinical Surgery, Cornell Medical College; Associate Attending Surgeon, St. Luke's Hospital; Attending Surgeon for Pediatrics, N. Y. Nursery and Child's Hospital.

1919. Speed, Kellogg, Sc.B., M.D., 122 South Michigan Avenue, Chicago, Ill. Clinical Professor of Surgery, Rush Medical College of University of Chicago; Attending Surgeon, Presbyterian Hospital.

1926. Stone, Harvey Brinton, M.D., 18 West Franklin Street, Baltimore, Md. Associate Professor of Surgery, Johns Hopkins Medical School; Visiting Surgeon, Johns Hopkins Hospital; Visiting Surgeon, Church Home and Infirmary, Union Memorial Hospital, Hospital for the Women of Maryland, Bon Secours Hospital and Mt. Sinai Hospital.

1923. St. John, Fordyce Barker, Sc.B., M.D., *Treasurer,* 180 Fort Washington Avenue, New York City. Associate Professor of Surgery, College of Physicians and Surgeons; Visiting Surgeon to the Presbyterian Hospital.

1936. Sturgeon, Charles T., M.D., 1930 Wilshire Boulevard, Los Angeles, Cal. Clinical Professor of Surgery, University of Southern California School of Medicine Senior Attending Surgeon, Los Angeles General Hospital; Director, Good Hope Hospital Association.

1920. †Taylor, Alfred S., P.B., A.M., M.D., 115 West Fifty-fifth Street, New York. Consulting Surgeon, Fordham Hospital; Consulting Neurological Surgeon, St. Luke's Hospital; Memorial Hospital, New York City; Glen Cove Hospital, Long Island; Tarrytown Hospital; Overlook Hospital, Summit, N. J.; Visiting Surgeon Neurological Institute of New York.

1916. †Terry, Wallace I., Sc.B., M.D., 2712 Broadway, San Francisco, Calif. Clinical Professor of Surgery, University of California.

1920. Tinker, Martin B., Sc.B., M.D., Savings Bank Building, Ithaca, N. Y. Surgeon to the Ithaca City Hospital and the Cornell Infirmary.

1932. Toland, Clarence G., M.D., 805 South Bonnie Brae, Los Angeles, Calif. Surgeon, Good Samaritan Hospital, Los Angeles Hospital, and St. Vincent Hospital, Los Angeles; Professor Clinical Surgery, School of Medicine, University of Southern California.

1928. †Torek, Franz, A.M., M.D., 91 Lloyd Road, Montclair, N. J. Consulting Surgeon and formerly Attending Surgeon to the New York Skin and Cancer Hospital; Consulting Surgeon to Lenox Hill Hospital; formerly Attending Surgeon to the Lenox Hill, Post-Graduate and St. Mark's Hospitals, and Adjunct Professor of Surgery, Post-Graduate Hospital.

1925. Trout, Hugh Henry, M.D., Jefferson Hospital, Roanoke, Va. Surgeon-in-Chief, Jefferson Hospital.

1918. Truesdale, Philemon Edwards, M.D., 151 Rock Street, Fall River,

ELECTED

Mass. Surgeon to St. Anne's and Highland Hospitals; Consulting Surgeon to Fall River City Hospital.

1928. Van Beuren, Frederick T., Jr., A.B., M.D., 65 Fifth Avenue, New York City, N. Y. Associate Dean, College of Physicians and Surgeons of Columbia University; Associate Professor of Clinical Surgery, College of Physicians and Surgeons; Attending Surgeon to Sloan Hospital for Women; Associate Visiting Surgeon to the Presbyterian Hospital.

1910. †Vander Veer, Edgar Albert, Ph.B., M.D., 28 Eagle Street, Albany, N. Y. Attending Surgeon, St. Peter's Hospital, Albany, N. Y.; Consulting Surgeon, Memorial Hospital, Albany, N. Y., Benedictine Hospital, Kingston, N. Y., Champlain Hospital, Plattsburgh, N. Y., and Greene County Memorial Hospital.

1902. †Vaughan, George Tully, M.D., LL.D., 1718 I Street, Washington, D. C. Professor of Principles and Practice of Surgery and Head of the Surgical Department, Georgetown University; Chief Surgeon, Georgetown University Hospital; Surgeon, Tuberculosis Hospital; Consulting Surgeon, Government Hospital for the Insane, Washington, D. C., U. S. Public Health Service and Washington Asylum Hospital; Commander, M.C., Auxiliary Navy, U.S.A.

1921. †Vaughan, J. Walter, A.B., M.D., 7815 East Jefferson Avenue, Detroit, Mich. Associate Attending Surgeon to Harper Hospital; Surgeon, Diagnostic Hospital.

1920. Verdi, William Francis, A.M., M.D., 27 Elm Street, New Haven, Connecticut. Clinical Professor of Surgery, Yale University School of Medicine; Chief Surgeon, Hospital of St. Raphael; Attending Surgeon, New Haven Hospital; Consulting Surgeon, St. Vincent's Hospital, Bridgeport; St. Mary's Hospital, Waterbury; Meriden Hospital, Meriden, Griffin Hospital, Derby. *Vice-President*, 1932.

1922. †Vincent, Beth, A.B., M.D., 925 Boylston Street, Boston, Mass. Chief of East Surgical Service, Massachusetts General Hospital; Consulting Surgeon, Infants' Hospital, Boston.

1933. Walker, Irving J., A.B., M.D., 520 Commonwealth Avenue, Boston, Mass., Surgeon in Chief, Harvard Surgical Teaching Service, Boston City Hospital; Clinical Professor of Surgery, Harvard Medical School.

1909. †Walker, John B., A.B., M.D., Sc.D., D.S.M., 117 East Seventy-second Street, New York City. Consulting Surgeon to Bellevue Hospital and Hospital for the Ruptured and Crippled, New York; Professor of Clinical Surgery, College of Physicians and Surgeons, Columbia University.

1932. Walters, Waltman, B.S., M.D., M.S. in Surgery, Mayo Clinic, Rochester, Minn. Associate Professor of Surgery, University of Minnesota (Mayo Foundation).

1933. Wangensteen, Owen H., M.D., 145 Melbourne Avenue, S. E., Minneapolis, Minn. Professor and Head of the Department of Surgery, University of Minnesota; Surgeon-in-Chief, University Hospital, Minneapolis, Minn.

1896. †Watson, Francis Sedgwick, A.B., M.D., South Dartmouth, Mass. Surgeon to the Boston City Hospital; Instructor in Genito-urinary Surgery, Harvard Medical School.

1913. †Watts, Stephen H., A.M., M.D., LL.D., "Broomley", University, Va. Former Professor of Surgery and Gynecology in the University of Virginia; Surgeon-in-Chief, University of Virginia Hospital,

ELECTED

1921. WHIPPLE, ALLEN O., B.S., M.D., Sc.D., Presbyterian Hospital, 620 West One Hundred and Sixty-eighth Street, New York City. Professor of Surgery, Columbia University; Director of Surgical Service, Presbyterian Hospital.

1926. †WHITTEMORE, WYMAN, Sc.B., M.D., 199 Beacon Street, Boston, Mass. Visiting Surgeon, Massachusetts General Hospital; Consulting Surgeon, Massachusetts Eye and Ear Infirmary, Sturdy Memorial, Peterboro and Beth Israel Hospitals.

1934. WILLARD, DE FOREST PORTER, B.S., M.D., 1726 Spruce Street, Philadelphia, Pa. Professor of Orthopaedics (Vice-Dean) Graduate School of Medicine, University of Pennsylvania; Orthopaedic Surgeon to the Philadelphia Orthopaedic Hospital and to the Hospital of Contagious Diseases; Consultant Orthopaedist to the Abington; the Bryn Mawr, the Chestnut Hill, the Shriners Hospitals and to the Delaware Hospital of Wilmington, Del.

1921. †WILLIAMS, HUGH, A.B., M.D., 301 Beacon Street, Boston, Mass. Assistant, Harvard Graduate School of Medicine; Visiting Surgeon, Massachusetts General Hospital.

1914. †WINSLOW, RANDOLPH, A.B., M.D., A.M., LL.D., 1900 Mt. Royal Terrace, Baltimore, Md. Emeritus Professor of Surgery, University of Maryland.

1935. WISE, WALTER DENT, M.D., F.A.C.S., 1120 St. Paul Street, Baltimore, Md. Professor of Clinical Surgery, University of Maryland and College of Physicians and Surgeons; Chief Surgeon, Mercy Hospital; Visiting Surgeon, Union Memorial Hospital, Hospital for the Women of Maryland, and South Baltimore General Hospital.

1931. WOLFER, JOHN A., M.D., 8 South Michigan Avenue, Chicago, Ill. Associate Professor, Northwestern University, Department of Surgery; Attending Surgeon Passavant Memorial Hospital; Supervising Consultant, Surgical Division, Veteran's Administration Hospital, Hines, Ill.

1918.† WOOD, ALFRED CONARD, M.D., 2035 Walnut Street, Philadelphia, Pa. Formerly Assistant Surgeon, University of Pennsylvania; Surgeon to the University, Philadelphia, Howard and Rush Hospitals; Consulting Surgeon to the Philadelphia Hospital, Montgomery Hospital, State Hospital for the Insane for Eastern Pennsylvania, Coatesville Hospital and Kent County General Hospital.

1901. †WOOLSEY, GEORGE, A.B., M.D., 117 East Thirty-sixth Street, New York City. Emeritus Professor of Clinical Surgery, Cornell University Medical College; Consulting Surgeon to Bellevue Hospital, Memorial Hospital, New York and the Infirmary for Women and Children.

1933. WRIGHT, ARTHUR MULLIN, M.D., 115 East Sixty-first Street, New York City. George David Stewart Professor of Surgery, New York University and Bellevue Hospital Medical College; Director Third Surgical Division, Bellevue Hospital; Director of Surgery, French Hospital; Consulting Surgeon, St. Vincent's Hospital, N. Y.; Mt. Vernon Hospital, Mt. Vernon; South Side Hospital, Bay Shore, N. Y.; St. Joseph's Hospital, Yonkers, N. Y.

1916. YATES, J. L., PH.B., Sc.B., M.D., 425 East Wisconsin Avenue, Milwaukee, Wis.

1935. ZIMMERMANN, HARRY BERNARD, M.D., 1360 Lowry Medical Arts Building, St. Paul, Minn. Assistant Professor of Surgery, University of Minnesota; Attending Surgeon, Charles T. Miller Hospital; Consulting Surgeon to the Shrine Hospital for Crippled Children.

SENIOR FELLOWS OF THE ASSOCIATION

Balch, Franklin G., A.M., M.D., 279 Clarendon Street, Boston, Mass.
Bevan, Arthur Dean, M.D., 122 South Michigan Avenue, Chicago, Ill.
Blake, John Babst, A.M., M.D., 311 Beacon Street, Boston, Mass.
Blake, Joseph A., A.B., Ph.B., M.A. (Hon.), M.D., Agua Caliente Ranch, Tucson, Arizona.
Brewer, George E., A.M., M.D., LL.D., F.R.C.S. (Ireland), 151 East Seventy-ninth Street, New York City.
Brewster, George W. W., A.B., M.D., 213 Beacon Street, Boston, Mass.
Brinsmade, W. B., A.B., M.D., Bedford Hills, N. Y.
Bruce, Herbert A., M.D., L.R.C.P. (London), F.R.C.S. (Eng.), 64 Bloor Street East, Toronto, Canada.
Brunn, Harold, M.D., 384 Post Street, San Francisco, Cal.
Buchanan, J. J., A.M., M.D., Ph.D., 1409 North Highland Ave., Pittsburgh, Pa.
Carrel, Alexis, L.B., Sc.B., M.D., Sc.D., Rockefeller Institute for Medical Research, New York City.
Clark, Edmund D., M.D., 23 East Ohio Street, Indianapolis, Ind.
Codman, Ernest Amory, 227 Beacon Street, Boston, Mass.
Cotton, Frederick J., A.B., M.D., A.M., 520 Commonwealth Avenue, Boston, Mass.
Crile, George W., A.B., M.D., Ph.D., LL.D., F.R.C.S. (Eng.), 2020 East Ninety-third Street, Cleveland, Ohio.
Cushing, Harvey, A.M., Sc.D., M.D., LL.D., F.R.C.S., Yale Medical School, New Haven, Conn.

Davis, John Staige, Ph.B., A.M., M.D., Severn Apartments, Baltimore, Md.
Davis, Lincoln, A.B., M.D., 279 Beacon Street, Boston, Mass.
Downes, William A., M.D., 424 Park Avenue, New York City, N. Y.

Eastman, Joseph Rilus, A.M., Sc.B., M.D., 970 North Meridan Street, Indianapolis, Ind.
Eberts, Edmond Melchior, M.D., M.R.C.S., (Eng.), L.R.C.P. (London), 1414 Drummond Street, Montreal, Canada.
Eliot, Ellsworth, Jr., M.D., 34 East Sixty-seventh Street, New York.
Elting, Arthur W., A.B., M.D., LL.D., 119 Washington Avenue, Albany, N. Y.
Estes, William Lawrence, A.M., M.D., Bethlehem, Pa.
Eve, Duncan, A.M., M.D., Eve Building, Nashville, Tenn.

Finney, John M. T., M.D., 3119 St. Paul Street, Baltimore, Md.
Fisher, William A., M.D., 715 Park Avenue, Baltimore, Md.

Gage, Homer, A.M., M.D., 72 Pearl Street, Worcester, Mass.
Gibbon, John H., M.D., 1608 Spruce Street, Philadelphia, Pa.
Gibson, Charles L., M.D., 1 West Fifty-fourth Street, New York, N. Y.
Greenough, Robert B., A.B., M.D., 8 Marlborough Street, Boston, Mass.

Hartwell, John A., Ph.B., M.D., 27 East Sixty-third Street, New York, N. Y.
Hodge, Edward Blanchard, A.B., M.D., 2019 Spruce Street, Philadelphia, Pa.
Hutchinson, James P., M.D., Media, Pa.

Jones, Daniel Fiske, A.B., M.D., 195 Beacon Street, Boston, Mass.
Jopson, John Howard, M.D., Waverly Mills, S. C.

Lilienthal, Howard, A.B., M.D., 52 East Eighty-second Street, New York City, N. Y.

McGuire, Stuart, M.D., LL.D., 1000 West Grace Street, Richmond, Va.
MacLaren, Murray, A.B., M.B., C.M., M.D., M.R.C.S. (Eng.), C.M.G., C.O.A., LL.D., M.P., 75 Coburg Street, St. John's, N. B., Canada.
Martin, Edward, A.M., M.D., LL.D., D.Sc., Media, Pa.
Martin, Walton, Ph.B., M.D., 230 East Forty-ninth Street, New York City, N. Y.
Matas, Rudolph, Sc.D., M.D., LL.D., F.R.C.S. (Hon. Eng.), 2255 St. Charles Avenue, New Orleans, La.
Mayo, William J., A.M., M.D. (Dublin; Cuba), Sc.D., LL.D., F.R.C.S. (Edin., England and Ireland), 701 Fourth Street, S. W., Rochester, Minnesota.
Mitchell, Charles F., M.D., 2003 Pine Street, Philadelphia, Pa.
Mitchell, James F., A.B., M.D., 1344 Nineteenth Street, Washington, D. C.
Murphy, Fred T., A.B., A.M., M.D., Penobscot Building, Detroit, Mich.

Neilson, Thomas R., M.D., 1937 Chestnut Street, Philadelphia, Pa.

Oliver, J. C., M.D., 628 Elm Street, Cincinnati, Ohio.
Olmsted, Ingersoll, M.B., 215 James Street, South, Hamilton, Ontario.

Plummer, Samuel C., A.B., M.D., 5844 Stony Island Avenue, Chicago, Ill.
Primrose, Alexander, C.B., M.B., C.M. (Edin.), F.R.C.S. (Eng.), LL.D. (Edin.), 518 Medical Arts Building, Toronto, Canada.

Richardson, Edward P., A.B., M.D., Massachusetts General Hospital, Boston, Mass.
Rixford, Emmet, B.S., M.D., 1795 California Street, San Francisco, Calif.
Rowan, Charles J., A.B., M.D., 702 Twenty-sixth Street, Santa Monica, Calif.

Scudder, Charles L., A.B., Ph.B., M.D., 144 Commonwealth Avenue, Boston, Mass.
Sellig, M. G., A.B., M.D., 4952 Park View Place, St. Louis, Mo.

Taylor, Alfred S., P.B., A.M., M.D., 115 West Fifty-fifth Street, New York City.
Terry, Wallace I., Sc.B., M.D., 2712 Broadway, San Francisco, Calif.
Torek, Franz, A.M., M.D., 91 Lloyd Road, Montclair, N. J.

Vander Veer, Edgar Albert, Ph.B., M.D., 28 Eagle Street, Albany, N. Y.
Vaughan, George Tully, M.D., LL.D., 1718 I Street, Washington, D. C.
Vaughan, J. Walter, A.B., M.D., 7815 East Jefferson Avenue E., Detroit, Mich.
Vincent, Beth, A.B., M.D., 925 Boylston Street, Boston, Mass.

Walker, John B., A.B., M.D., Sc.D., D.S.M., 117 East Seventy-second Street, New York City.
Watson, Francis S., A.B., M.D., South Dartmouth, Mass.
Watts, Stephen H., A.M., M.D., LL.D., "Broomley", University, Va.
Whittemore, Wyman, Sc.B., M.D., 199 Beacon Street, Boston, Mass.
Williams, Hugh, A.B., M.D., 301 Beacon Street, Boston, Mass.
Winslow, Randolph, A.B., M.D., A.M., LL.D., 1900 Mt. Royal Terrace, Baltimore, Md.
Wood, Alfred Conard, M.D., 2035 Walnut Street, Philadelphia, Pa.
Woolsey, George, A.B., M.D., 117 East Thirty-sixth Street, New York City.

HONORARY FELLOWS OF THE ASSOCIATION

ELECTED

1919. Bastianelli, Raffaele, Via dei Villini, 2, Rome, Italy; Consulting Surgeon of the United Hospitals of Rome; Associated Professor of Surgery, Royal University, Rome, and Director of the Institute "Regina Elena" for Tumors Research.

1922. Chutro, Dr. Pedro, Junin, 1476, Buenos Aires, Argentina. Professor of Surgery, University of Buenos Aires; Honorary Professor, University of Madrid, Spain; Fellow, Royal Academy of Medicine of Spain, of the Académie de Médecine de Paris, of the Surgical Association of Buenos Aires, of the Société de chirurgie de Paris; Honorary Fellow, Association of Military Surgeons of the United States.

1911. von Eiselberg, Professor Anton, I. Molkerbastei 5, Vienna, Austria. Professor of Surgery, Vienna University.

1933. Fraser, John, 32 Moray Place, Edinburgh, Scotland.

1919. Hartmann, Dr. Henri, Hon. F.R.C.S. (Eng.), 4 Place Malesherbes, Paris, France. Professeur de Clinique Chirurgicale, Chirurgien de l'Hôtel Dieu. Membre de l'Académie de Médecine.

1933. Ireland, M. W., M.D., A.M., LL.D., R.C.S. (Edinburgh), Major General, United States Army, Retired, Chief Surgeon American Expeditionary Forces, Surgeon General United States Army, 1918–1931. Past President American College of Surgeons. Member National Board of Medical Examiners. Address: 1870 Wyoming Avenue, N.W., Washington, D. C.

1918. Keogh, Sir Alfred, Hy., G.C.B., Hon. F.R.C.S., c/o Sir W. M. Russell, K.C.M.G., Merwald House, Upper Walmer, Kent, England.

1922. Leriche, Dr. René, 19 rue Jean Jacques Rousseau, Strasbourg, France. Professeur de Clinique Chirurgicale à la Faculté de Médecine de Strasbourg.

1927. de Martel, Thierry, M.D., 18 Rue Weber, Paris, XVI°, France. Chief Surgeon, American Hospital of Paris; Surgeon to Texel, Heine-Fould and Private Surgical Hospitals; Honorary Consultant, Society of Clinical Surgery, Paris.

1907. Myles, Sir Thomas, M.D., F.R.C.S.I., 33 Merrion Square N., Dublin, Ireland. Past President of Royal College of Surgeons, of Ireland.

ELECTED

1930. Nyström, Gunnar, M.D., Uppsala, Sweden. Professor of Surgery, University of Uppsala, Sweden. University Hospital Surgical Clinic, Uppsala.

1925. Power, Sir d'Arcy, K.B.E., A.M., M.B. (Oxon.), F.R.C.S. (Eng.), 10 A. Chandos Street, London, England. Consulting Surgeon, St. Bartholomew's Hospital, Honorary Librarian and formerly Vice President, R. C. S., Late President of the Bibliographical Society.

1912. Stiles, Sir Harold Jalland, K.B.E., LL.D., D.Sc., M.B., F.R.C.S. (Edin.), F.A.C.S., F.R.C.S.I., 9 Great Stuart Street, Edinburgh, Scotland. Formerly Regius Professor of Clinical Surgery, University of Edinburgh; Past President of the Royal College of Surgeons of Edinburgh; Consulting Surgeon to the Edinburgh Royal Infirmary, Chalmers Hospital, and the Royal Edinburgh Hospital for Sick Children, Edinburgh.

1920. Wallace, Sir Cuthbert, K.C.M.G., C.B., D.S.M. (U. S. A.), M.B., B.S. (London), F.R.C.S. (Eng.), 5, Cambridge Terrace, Regent's Park, N. W. 1, London, Eng.; Consulting Surgeon to St. Thomas's Hospital, London; Formerly Dean of the Medical School; Director, Mt. Vernon Hospital, London.

1930. Young, Archibald, B.Sc.; M.B., C.M.; F.R.F.P.S. Glas.; F.A.C.S. (Hon.); Docteur, Hon. Causa (Strasbourg); 5 Park Gardens, Glasgow, C 3. Regius Professor of Surgery, University of Glasgow; Visiting Surgeon, Western Infirmary, Glasgow; Hon. Member, Academy of Medicine, Rome; Hon. Corresp. Member, Soc. Med. Serv., Lisbon; Member, Association Française de Chirurgie; etc. Past President of the Association of Surgeons of Great Britain and Ireland (1927); Past President of the Royal Med. Chir. Society of Glasgow; Past Professor of Surgery, and Dean of the Med. School, The Anderson College of Medicine, Glasgow; etc.

DECEASED FELLOWS OF THE ASSOCIATION

Name	Date of death
Abbe, Robert	March 7, 1928
Agnew, D. Hayes	March 22, 1892
Alexander, Emory Graham	August 29, 1930
Allen, Dudley Peter	January 6, 1915
Allis, Oscar Huntington	May 16, 1921
Andrews, E. Wyllys	January 21, 1927
Annandale, Thomas	1907
Armstrong, George E.	May 25, 1933
Ashhurst, Astley Paston Cooper	September 19, 1932
Ashhurst, John, Jr.	July 7, 1900
*Atlee, John Light	October 1, 1885
*Ballance, Sir Charles Alfred	February 8, 1936
Ballin, Max	March 3, 1934
Barrow, David	August 18, 1932
Barton, James M.	June 28, 1926
Beckman, Emil Hessel	November 7, 1916
Benham, Silas Nelson	November 3, 1890
*von Bergmann, Ernst	Died since 1906 Meeting
*Bigelow, Henry Jacob	October 30, 1890
*Billings, John Shaw	March 11, 1913
*Billroth, Albert Christian Theodor	January 5, 1894
†Binney, John Fairbairn	November 28, 1936
Bloodgood, Joseph Colt	October 27, 1935
Bolling, Richard A.	April 6, 1929
Bontecou, R. B.	1906-1907
Bosher, Lewis C.	September 12, 1920
Bottomley, John T.	December 17, 1925
*Bowlby, Sir Anthony	April 9, 1929
Bradford, Edward Hickling	May 7, 1926
Briggs, William Thompson	June 13, 1894
Brinton, John Hill	March 18, 1908
Bristow, Algernon Thomas	March 26, 1913
Brock, Hugh W.	April 24, 1882
Bryant, Joseph Decatur	April 7, 1914
*Bryant, Thomas	1914-1915
Bull, William Tillinghast	February 22, 1909
Bunts, Frank E.	November 26, 1928
Burrell, Herbert Leslie	1910
Byrd, William Andrew	August 14, 1887
Cabot, Arthur Tracy	November 4, 1912
Campbell, Henry Fraser	December 15, 1891
Campbell, William F.	September 7, 1926
Carmalt, William H.	1928
Carson, Norman Bruce	August 29, 1931
Cheever, David Williams	December 27, 1915
*Chiene, John	May 29, 1923
Cole, Charles Knox	February 27, 1920
Coley, William B.	April 16, 1936
Conner, Phineas Sanborn	March 26, 1909

† Senior. * Honorary.

Name	Date of death
Connors, John F.	January 5, 1935
Cowan, John Francis	1929
Cunningham, Francis Deane	September 11, 1885
Cushing, Hayward Warren	May 8, 1934
*Czerny, Prof. Dr. Vincenz	October 3, 1916
Da Costa, John Chalmers	May 17, 1933
Dandridge, Nathaniel P.	November 6, 1910
Davis, Gwilym G.	June 16, 1918
Davis, John Staige	July 18, 1885
Dawson, William Wirt	February 16, 1893
Deaver, Harry C.	June 25, 1931
Deaver, John B.	September 25, 1931
Delatous, H. Beeckman	August 23, 1930
Dennis, Frederic Shepard	March 8, 1934
*Depage, Antoine	June 9, 1925
Dowd, Charles N.	May 24, 1931
Dugas, Louis Alexander	October 19, 1884
Dunn, James Henry	June 16, 1904
Dunott, Thomas Justus	May 20, 1893
*Durham, Arthur Edward	May 7, 1895
Elliott, John Wheelock	September 17, 1925
*Erichsen, Sir John Eric	September 23, 1896
*von Esmarch, J. Friederich	February 23, 1908
Fenger, Christian	March 7, 1902
Ferguson, Alexander Hugh	October 20, 1911
Forbes, William Smith	December 17, 1905
Fowler, George Ryerson	February 6, 1906
Frazier, Charles H.	July 26, 1936
Freeman, Leonard	December 27, 1935
*Friedrich, Paul Leopold	January 5, 1916
Gaston, James McFadden	November 17, 1903
Gay, George Washington	May 30, 1931
Gerrish, Frederic Henry	September 8, 1920
Gerster, Arpad G.	March 11, 1923
*Gorgas, William C.	July 4, 1920
Greene, W. Warren	September 10, 1881
Gross, Samuel David	May 6, 1884
Gross, Samuel Weissel	April 16, 1889
Gunn, Moses	November 4, 1887
*Gussenbauer, Karl	June 19, 1903
Guthrie, George Washington	June 1, 1915
Halstead, Albert E.	December 6, 1926
Halsted, William Stewart	September 7, 1922
Hamann, Carl A.	July 12, 1930
Harrington, Francis B.	June 8, 1914
Harris, Malcolm L.	March 22, 1936
*Harrison, Reginald	February 28, 1908
Harte, Richard H.	November 14, 1925
Hearn, W. Joseph	December 20, 1917
Hedblom, Carl A.	June 6, 1934
Hodge, H. Lenox	June 16, 1881
Hodgen, John T.	April 28, 1882
Homans, John	February 7, 1903
*Horsley, Sir Victor Alexander, C.B.	July 16, 1916

* Honorary.

Name	Date of death
Horwitz, Orville	January 28, 1913
*Horwitz, Phineas J.	September 28, 1904
Hotchkiss, Lucius Wales	April 11, 1926
Hubbard, Joshua Clapp	January 11, 1934
Hughes, J. C.	August 10, 1881
*Humphrey, Sir George Murray	September 24, 1896
*Hunt, William	January 5, 1897
Huntington, Thomas W.	April 19, 1929
Hupp, Frank LeMoyne	December 26, 1929
Hutchinson, James A.	June 30, 1929
Jackson, Jabez N.	March 18, 1935
Jacobson, Nathan	September 16, 1913
Johnson, Alexander B.	September 4, 1917
Johnson, Robert W.	November 13, 1930
Johnston, Christopher	December 20, 1916
Johnston, George Ben	October 11, 1916
Jonas, August Frederick	November 12, 1934
Judd, Edward Starr	November 30, 1935
Kammerer, Frederic	September 26, 1928
**Keen, William Williams	June 7, 1932
King, Alfred	June 4, 1916
Kinloch, Robert Alexander	December 23, 1891
Klopp, Edward J.	September 19, 1936
*Kocher, Theodor	July 27, 1917
Lane, Levi Cooper	February 18, 1902
Law, Arthur Ayer	July 9, 1930
*von Langenbeck, Bernhardt	September 30, 1887
Le Conte, Robert G.	August 5, 1924
Lee, Burton J.	November 12, 1933
Levis, Richard J.	November 12, 1890
*Lister (Lord), Joseph	February 11, 1912
Little, James Lawrence	April 4, 1885
*Lorthioir, Jules	1931
Lothrop, Howard Augustus	June 4, 1928
Lutz, Frank Joseph	March 24, 1916
McArthur, L. L.	November 5, 1934
McCann, James	June, 1893
McCosh, Andrew J.	December 3, 1908
McGraw, Theodore A.	September 6, 1921
McGuire, Edgar R.	April 22, 1931
McGuire, Hunter	September 19, 1900
McMurtry, Lewis Samuel	February 1, 1924
McRea, Floyd W.	August 13, 1921
McWilliams, Clarence A.	January 20, 1927
*MacCormac, Sir William	December 4, 1901
MacDonald, Willis Goss	December 30, 1910
*Macewen, Sir William	March 22, 1924
Mackenzie, Kenneth Alexander Jas.	March 15, 1920
MacLaren, Archibald	October 12, 1924
MacMonagle, Beverly	May 22, 1912
*Makins, Sir George	November 2, 1933
Markoe, Francis Hartman	September 13, 1907
Marks, Solon	September 29, 1914
Mathews, Francis Stuart	February 17, 1936
*Maunoury, Gabriel	December, 1925

** Original and Honorary. * Honorary.

Name	Date of death
Mason, James Tate	June 20, 1936
Mastin, Claudius Henry	October 3, 1898
Mastin, William McDowell	February 3, 1933
*Mayo-Robson, Sir Arthur	October 12, 1933
Mears, J. Ewing	May 28, 1919
Meyer, Willy	February 25, 1932
Michael, J. Edwin	December 7, 1895
*Mikulicz-Radecki, John V.	June 17, 1905
Miles, Albert B.	August 5, 1894
Millard, Perry H.	February 1, 1897
Miller, Truman W.	May 31, 1900
Mixter, Samuel Jason	January 19, 1926
Monks, George Howard	January 26, 1933
*Moore, Edward Mott	March 3, 1902
Moore, James E.	November 2, 1918
Moschcowitz, Alexis V.	December 21, 1933
*Moynihan, Berkeley George Andrew	September 7, 1936
Mudd, Harvey G.	August 16, 1933
Mudd, Henry Hodgen	November 20, 1918
Mumford, James Gregory	October 18, 1914
Munro, John C.	December 6, 1910
Murphy, John B.	August 11, 1916
Murray, Francis W.	April 12, 1929
de Nancrède, C. B. G.	April 12, 1921
Nichols, Edward Hall	July 6, 1922
Norris, Basil E.	October, 1895
*von Nussbaum, J. N.	October 31, 1890
Ochsner, A. J.	July 25, 1925
*Ollier, Leopold	November 26, 1900
Oviatt, Charles W.	October 31, 1912
Owens, John E.	December 21, 1922
*Paget, Sir James	December 30, 1899
Pancoast, William H.	January 5, 1897
Parham, Frederick W.	March 6, 1927
Park, Roswell	February 15, 1914
*Parker, Willard	April 25, 1884
Parkes, Charles T.	March 28, 1891
Parkhill, Clayton	January 16, 1902
Parmenter, John	June 1, 1932
*Péan, Jules E.	January 30, 1898
Peck, Charles H.	March 28, 1927
Peck, Washington F.	December 30, 1891
Pilcher, Lewis Stephen	December 24, 1934
Pilcher, Paul Monroe	January 4, 1917
Porter, Charles Allen	July 3, 1931
Porter, Charles Burnham	May 21, 1909
Porter, Miles Fuller	December 6, 1933
Porter, William Gibbs	January 30, 1906
Powers, Charles A.	December 23, 1922
*Pozzi, Jean Samuel	May, 1918
Prewitt, Theodore F.	October 17, 1904
Prince, David	December 19, 1889
Ransohoff, Joseph	March 10, 1921
Reed, Thomas Baird	April 1, 1891
Richardson, Maurice Howe	July 31, 1912
Richardson, Tobias Gibson	May 26, 1892

* Honorary.

Name	Date of death
Roberts, John B.	November 28, 1924
Rodman, William Louis	March 8, 1915
Ross, George Gorgas	December 27, 1922
Rushmore, John D.	March 23, 1929
Sabine, Thomas Taunton	August 23, 1888
Sayre, Lewis A.	September 21, 1900
*Schede, Max	January, 1903
Senn, Nicholas	January 2, 1908
Sheppard, Francis J.	January 18, 1929
Sherman, Harry M.	May 15, 1921
Sherwood, Walter A.	November 21, 1931
*Sims, J. Marion	November 13, 1883
Sistrunk, Walter E.	March 6, 1933
Smith, Rea	November 29, 1935
*Smith, Stephen	August 26, 1922
Souchon, Edmond	August 5, 1924
Speese, John	October 15, 1933
Squire, Truman Hoffman	November 27, 1889
Starr, Clarence Leslie	December 25, 1928
Starr, Frederic Newton Gisborne	April 21, 1934
Stewart, Francis T.	February 4, 1920
Stewart, George David	March 9, 1933
Stillman, Stanley	
Summers, John Edward	February 7, 1935
*Taylor, Sir William	January 29, 1933
Taylor, William J.	January 22, 1936
*Terrier, Louis Felix	April 9, 1908
*Thiersch, Karl	April 28, 1895
Thompson, James E.	April 8, 1927
Thompson, Joseph Ford	February 1, 1917
Thompson, Joseph W.	March 30, 1886
*Thomson, Alexis	March 4, 1924
Thomson, William	August 3, 1907
Tiffany, Louis McLane	October 23, 1916
*Tuffier, Theodore	October 27, 1929
Vander Veer, A.	December 19, 1929
Varick, Theodore Romeyn	November 23, 1887
*Verneuil, Aristide A. S.	June 11, 1895
*von Volkmann, Richard	November 28, 1889
Wainwright, Jonathan	August 3, 1934
Walker, Edward W.	October 7, 1925
*Walther, Charles	December 1, 1935
Warren, John Collins	November 3, 1927
Watson, Beriah Andrew	December 22, 1892
Weeks, Stephen Holmes	September 1, 1909
Weir, Robert F.	April 6, 1927
Weist, Jacob Rowland	May 14, 1900
*Wells, Sir Thomas Spencer	January 31, 1897
Wharton, Henry R.	December 3, 1925
White, James William	April 24, 1916
Wight, Jarvis S.	November 16, 1901
Willard, DeForest	October 14, 1910
Wood, James R.	May 4, 1882
*Yandell, David Wendell	May 2, 1898

* Honorary.

ADDRESS OF THE PRESIDENT

THE MAKING OF A SURGEON

EUGENE H. POOL, M.D.
NEW YORK

THIS is my first opportunity to express appreciation of the great honor you have conferred upon me by electing me president of the American Surgical Association. Being unexpected, it is the more welcome.

Instead of attempting the customary philosophic discourse, I have chosen as the subject of my address, "The Making of a Surgeon." It is popularly believed that a surgeon, like a poet or musician, is born, not made; but in reality every detail of his development is the result of long continued effort and concentrated purpose. He is an artificial, not a natural product. But it must not be inferred that extrinsic influences are wholly responsible for his development. His progress for the most part is dependent upon self-training. During the period of preparation, organized medical education is mainly responsible, but it becomes progressively less important as the specialized field is entered and followed. In the preparatory period the function of organized education is clear cut: in the period of specialization it is confused and uncertain; so much so that there is perennial discussion as to what should constitute the training of a surgeon.

Let us view the problem from the standpoint of him who is being trained. Let us put ourselves in his position, and travel along the paths which he must tread. In order to secure a contemporaneous viewpoint, rather than one in retrospect, I stimulated our house officers to analyze the problem, and to record the queries which arose during their discussions. We can perhaps profit by the friendly cogitations of this composite young surgical mind.

It is usually while he is still in school that a boy elects medicine as his profession. His reasons vary widely. He thinks, perhaps, that he may thereby secure an established social position; or he hopes by it to achieve substantial material reward: or he is influenced by the fact that he possesses some mechanical adroitness. Perhaps his father says of him, "Charlie can do anything with his hands. He has done all our repairs since he was eight years old." But the most universally controlling motive is humanitarian; an indefinite hope of contributing to the welfare of mankind. It may originate

in the memory and inspiration of the kindly old gentleman who drove out one stormy night to treat him for croup; a man who, he had observed—like Ian McLaren's Doctor MacLure—"did his best for the need of every man, woman and child in this straggling district, year in, year out, in the snow and in the heat, in the dark and in the light, without rest and without holiday for forty years. It was michty tae see him come: the varra look o' him was victory." So the boy elects medicine, without carefully weighing the duration and intensity of the struggle, without due consideration of the financial strain.

He enters college actuated by an ideal. For two to three and a half years he must devote much of his time to premedical courses. He does not appreciate what a medical career involves and in many instances it is only after years of effort that he finds the life is not what he anticipated. Would it not be well for his college at the outset to inform him as to the true conditions? Should not a kindly soul explain to all premedical students the struggle, competition, expense and doubtful rewards; not with the view of discouraging a serious aspirant, but as a matter of justice, and to prevent disappointment and waste?

In his college course the young man should receive a broad training. A thorough knowledge of English, philosophy, mathematics, economics, and the languages will, in the long run, be of more value than a smattering of biology and zoology. Should not sculpture and the art of drawing and painting be more highly regarded, combining as they do, dexterity with an esthetic outlook? "Colleges," said Emerson, "can only highly serve us when they aim not to drill but to create: when they gather from far every ray of various genius to their hospitable halls and by the concentrated fires set the hearts of their youth on flame." The premedical requirements appear to me too ambitious and too highly specialized, and for this opinion I find support in the Report of the Commission on Medical Education,* which emphasizes the lack of proper motivation of the premedical science training in many colleges, and states that much of the science teaching is presented from the special interest of the teacher or department. Organic chemistry, the Commission finds, is frequently taught from the standpoint of industrial uses, and much of the teaching of inorganic chemistry emphasizes its commercial applications. An adequate knowledge of the principles and methods of these sciences for the purposes of medical education could probably be secured in less time than is now required "if the courses were focussed upon the needs of the student." The objective should be a broad general education. An intellectual attitude, common sense, the knowledge of people, of the humanities, of one's self, will be more useful than premature specialized training in preparing the student for a successful and helpful career. Specialized training should come more slowly, and by absorption and election. Science is a strong draught which often intoxicates the immature mind. Its study should not be prejudicial to

* "The Report of the Commission on Medical Education" has been fully drawn upon in this discussion.

cultural development and the appreciation of the beauties of life. The development of a surgeon must be of many years' duration, and the early years must of necessity be lean and unproductive. Therefore the plan of education should be laid for a long race.

> "The heights by great men reached and kept
> Were not attained by sudden flight,
> But they, while their companions slept,
> Were toiling upward in the night."

My views in this respect are at variance with those commonly accepted and followed. Note, for instance, the system in England. A boy often has completed his premedical courses on entering the university; there, he customarily devotes all of his time to the medical sciences. He thus may embark on clinical work on quitting the university at 21 or 22 years of age. This is an extreme form of the early specialization which I condemn.

"What medical school shall I enter?" the enthusiastic young Sir Galahad asks. "I am at last on my own, with the world at my feet." But how hopes are to be shattered! Some months after his application, months of fretting and uncertainty, a curt message tells him that he has not been accepted by the school of his choice. In the leading schools only one in ten is taken. He must try, and perhaps keep on trying elsewhere, until at last he is privileged to enter a school. Although I recognize the great difficulties confronting the authorities in making their selections I fear there is not always the kindliness and judgment shown which should guide those who decide the destinies of young men. Every candidate should find a welcoming, not a repelling or suspicious atmosphere.

It is generally believed that "the type of student who studies medicine is determined by the professional opportunities and social recognition of the physician." While this is to some extent true, in the last analysis it is the instructors in the premedical courses of our colleges who select the future doctors of the country, because the medical colleges rely largely upon their recommendations in selecting candidates. It is a question whether these men recognize this responsibility and give sufficient thought to the qualifications of candidates beyond the question of marks. They should be instructed as to the importance of their influence and be made more cooperative. Where a medical school is associated with a university it would be advisable for its science instructors to participate in the premedical science courses in the college. This would not only be an inspiration to the young man but would also aid in the selection of the proper candidates.

The undergraduate surgical courses are pretty well standardized, and the same principles and policies prevail in most schools. A perusal of the remarkably comprehensive report of the Commission on Medical Education, published in 1932, shows what immense efforts are being made towards perfecting the undergraduate courses in our medical schools. It is well recognized that the fundamental purpose of medical education is to provide enough

competent men to meet the needs of the community for the care of the sick and the prevention and control of disease; and that the method should be to teach the man to think for himself, not, as of old, to cram his mind full of facts. There seems little of importance to criticize, although we still hear discussions as to details. It is often stated that anatomy has become slighted, yet it must be recognized that the weight of the curriculum as a result of the many added courses now makes it impossible to train all students, as of old, in the details of anatomy. Therefore, the surgical aspirant must supplement the routine courses by advanced anatomic studies, which should include the application of anatomy to surgery. The same may be said of physiology which, I think, is equally important. Pathology is perhaps being unwisely curtailed. Undue weight is placed on rare diseases, because they interest the instructor. It is more important to teach the differentiation of carcinoma of the rectum from hemorrhoids than to put the young man ever on the alert to recognize a sacrococcygeal chordoma. There does not appear to be sufficient coordination among the various departments in their teaching. Moreover, the pendulum has swung too far away from didactic teaching, and the lecture has received too severe an indictment. Personal contacts with mature experienced clinicians are neglected, and the young man of relative inexperience is given too prominent a place as a teacher. Experience, mellowness, stability from years of struggle and strife are not sufficiently appreciated and used. It were better for the student in his early years to be a hero worshiper than an iconoclast.

The medical degree is at last his, but this, he realizes, is only the beginning. He faces the problem of obtaining an internship, to learn the practical uses of his theoretical knowledge. It is fortunate that about 95 per cent of graduates obtain this opportunity. Graduate study then begins. In the case of surgery it seems obvious, though not generally accepted, that at some part of his training he should become thoroughly familiar with the human body and its general deviations from normal. This demands an intimate knowledge of internal medicine, psychiatry and pathology. At least one year on a medical service should be elected at the outset, special attention being directed to the heart and lungs. If delayed, it will never be done. The man thus gains a first hand working knowledge of the unit on which he is to do his life work. I maintain that successful surgery depends largely upon anticipating and avoiding troubles, and recognizing and treating complications in their incipiency. The surgeon, then, must be expert with the stethoscope and with general changes in the condition of a patient. He must have the mind and eye of an internist, the hand of a surgeon. The interests of the patient are best conserved if all cases are followed and treated by the internist and surgeon together. But this is possible only in institutional and group medicine. Therefore, the surgeon should be so equipped medically that he can recognize at the first signs that something is wrong, and pretty much what is wrong.

Quite as important as familiarity with the physical aspects of man is

some knowledge of his mental habits and peculiarities. The psychology of the patient, that is his susceptibility and responses to outside influences and personalities is not a new concept. About 1800 years ago Galen exhorted his pupils to be careful not to arouse and irritate the patient when entering the sickroom by stumbling and screaming. He gave them benevolent advice with respect to their clothing, their behavior and the conversation to be held with the patient. Cleanliness was recommended, and they were forbidden to eat onions or garlic or to drink too much wine before visiting the sick.

While every surgeon recognizes that his actions and words often have a profound influence on the patient, this is not sufficient. He should be familiar with the peculiarities of the mind which are classified under such terms as neurosis, psychosis and hysteria. We are all more or less unconsciously psychiatrists, but there should be a deliberate and planned effort to make the young man a good psychiatrist, and for this he should have formal training. A more general adoption of this principle would probably minimize the activities of the cults.

In 1927, rumors were heard that some of our surgical cases had landed in Bloomingdale Asylum; also that criticisms were expressed that operations had been performed upon these patients. This led us to arrange for a close association with Doctor Henry,* one of their leading psychiatrists. His findings, based on 300 psychiatric consultations, may be summarized. Fifteen per cent of the patients in general hospitals require psychiatric supervision. While you may think such a high percentage does not apply elsewhere than in New York, nevertheless the problem is a real one in every community. The study revealed the absence of psychiatric data in the histories, an unscientific attitude toward the psychiatric features in the patient's illness, and a laborious method of arriving at a diagnosis through a process of elimination. An average of seven days elapsed after admission to the hospital before there was a psychiatric consultation even in cases found to be uncomplicated by physical disease. A period of a week to a month passed before the patients were discharged. There seems to be a general misconception that in order to deal with psychiatric problems the physician must ever be on the watch for obscure psychic influences and lecherous fixations. This is without foundation.

Doctor Henry recommended that no medical student should be permitted to graduate without having had an elementary course in the psychopathic aspects of general hospital practice; and that a period of study in a psychopathic hospital should be a part of all hospital internships. With these recommendations I emphatically agree.

During his internship, as well as during his undergraduate days, the young man should be encouraged and directed in the use of the library, which should be complete and easy of access. The urge comes chiefly from exam-

* Some Aspects of Psychiatry in General Hospital Practice: G. W. Henry, Bloomingdale Hospital.

ple. We have all felt the stimulation of seeing Paget's works presented at a clinic. Another teacher brings from the library Duplay's original article to reveal the details of the lesion which is known by his name. While such examples could be multiplied, this practice is far too infrequent.

We might profit by Ruskin's observation, "That to use books rightly is to go to them for help, to appeal to them when our knowledge and power of thought fails: to be led by them into wider sight—purer conception—than our own, and receive from them the united sentence of the judges and councils of all time against our solitary and unstable opinion."

The problem as to an internship centers upon the type of service. In the average hospital the internship affords an admirable training in the fundamentals of surgery, though it does not make a surgeon; under the resident system the resident obtains a supertraining, the intern as a rule relatively little; indeed, it is said that the resident often makes the life of the intern one of misery. It is pretty well established that there is a place for both systems. However, the actual details as to their best practical application appear nowhere to have been perfected. The intern plan is traditional and has been in vogue since the earliest days of American hospitals. Radical changes seem indicated; to these we shall refer later.

With the resident system is closely linked the full time policy which in practice has offered certain advantages; notably, more supervision, teaching and research. But, whereas devised to correct an evil, it has in some instances become an evil.

Many surgical departments are now so large and many sided that there must be a full time director. But should the great contributor or research worker, who is as rare as the great composer, be obliged to spend his time on administration work? No! He should be carefully nurtured like a rare plant. It is as appropriate to put him in charge of the details of laboratories, museums, elementary teaching and questions of discipline, as to put a Wagner in charge of music in a girls' seminary.

While the administrative head of the surgical department in the larger institutions must devote his whole time to the service, it is problematic how far the full time principle should be extended beyond this. The question obviously has not been satisfactorily answered, since we see in every progressive institution frequent changes in its methods. My own conviction is that certain of the younger men should be on full time—Fellows, if you please—also the younger attendings, who might be allowed to practice in the institution. The upper group should be on part time. I believe that men who have struggled in the competition of practice, and have survived, are in general better qualified to tell the young man who is embarking on practice what are the problems, and how they should be met, than is the sequestered hermit who has never been out of a hospital or off a salary.

The discussion of the system, however, appears to be receiving more attention than it deserves. The interests of the institution, whether it be

hospital or school, depend upon the cooperation and confidence of the public and the outside profession. A broad minded generous policy under almost any method, equal professional ability being assumed, will spell success in teaching, care of the patient and clientele for the institution. A short sighted selfish policy will beget disloyalty, lack of confidence and failure. The results, therefore, depend more upon the individual than the method.

The intern should be taught to recognize that every surgeon should be impregnated with the tenets of pathology. Surgery and pathology have been closely linked from the beginning. As you must recall, Bichat, the father of modern histologic pathology, would probably have died unsung had it not been for his contact with Pierre Desault and the inspiration he received from that master surgeon. In recent developments surgery has been advanced quite as much by the microscope as by the knife.

I feel that the laboratories of a hospital should be the hub, intellectually and physically, from which all else radiates; and, reciprocally, that all paths should be planned to lead to them, so that in the course of his daily perambulations the clinician perforce finds himself in juxtaposition with the laboratories; that the laboratories be directed by a broad minded leader who will welcome the clinician and give willingly of his time. In a word, the laboratory should be the vitalizing force of the institution. Philosophy, erudition, research, encouragement, should there be found. The clinician without this stimulus will in general have a narrow limited viewpoint. On the other hand, such daily contacts will lead him from the narrow confines of pure clinicism (if I may coin a word) into the limitless expanses of scientific and philosophic thought. Intimate contacts should be encouraged; these are possible only by rubbing heads over the same eyepiece. If this attitude does not prevail, there is a tendency to become more and more of a technician and rule of thumb clinician with a narrowing in breadth and lowering of ideals.

Our young man—though now some 30 years of age—is at last faced with the problem of shifting for himself. What does he find? The field of medicine is overcrowded, if we may accept such an authority as the Commission on Medical Education. There is every reason to believe that the specialty of surgery is particularly overcrowded. It affords unusual attractions through its spectacular aspects, its rapid and clear cut results, its greater financial rewards.

The oversupply of physicians in this country, estimated at 25,000, is most marked in densely settled areas. There should be greater efforts to influence a larger number of recent graduates to enter the rural districts. They should be informed as to the needs and opportunities and even trained for such work as has been done in some of the smaller schools, as was strikingly presented to this Association by Elting of Albany.

The vast extension of knowledge and technical developments in recent years makes it impossible for any man to be experienced in all branches. Specialization has therefore become necessary, but it has been overdone, as

was emphasized by our former President, Doctor Jones. Twenty years ago the general surgeon performed tonsil and mastoid operations as well as gynecologic, urologic, neurologic and orthopedic surgery. It became evident that the welfare of the patient was not met by such widespread activities. Gradually the field has been subdivided, until now we have the hand, plastic, hernia, breast, rectal and thyroid specialist, and we may expect, if the tendency is not arrested, a further extension of specialism into even more minute subdivisions. Such extreme limitation is stultifying and unnecessary. Under these conditions the surgeon becomes almost exclusively a craftsman. Such a man cannot be a real teacher or director.

Some specialization is essential with the proviso that the specialist has had a preliminary general surgical training. We have all been called upon to remove a jugular vein after a mastoid operation and to do a jejunostomy after a hysterectomy, the specialist not trusting himself beyond his limited field; and how often are we called upon to correct the mistakes of the casual operator?

Our composite young mind, which suggested many of my topics, framed the question, "What place does research or experimental surgery occupy in the surgeon's training?" The answer calls for a subdivision of surgery; first, the purely clinical, which includes the casual operator, and second, those who are inherently investigative; the ones who will become notable teachers, trail-blazers and leaders. For the former, rule of thumb methods, obtained from the text-book, are satisfying; for the latter, everything seems imperfect; in them there is an ever present urge for improvement. The impulse is irresistible, and, as in philosophy, is the search for truth, not self-exploitation. Thus it depends upon the man. He who does research for self-exploitation had better leave it alone. He who does research from an impelling and irresistible impulse will profit greatly as a result of justifiable efforts.

I do not subscribe to the prevailing attitude in regard to research, that he who abuses a dog is necessarily a scientist and research worker, but he who performs worthily upon man is something to be despised; or that the laboratory is necessarily superior to the operating room as a means of learning to care for human ills and adding to our knowledge of man and his defects. No! Basic contributions are frequent through clinical observation and study, and often constitute research of a high grade.

> "Know then thyself, presume not God to scan:
> The proper study of mankind is Man."

Conditions of investigation or research now differ radically from the earlier days of medical progress. In the past, individuals pursued lines of investigation relatively unaided. Now, the important problems demand group effort. Take, for instance, the question of neoplasms. Vast progress certainly has been made during the fourscore years since modern pathology was initiated by Virchow's contribution on cellular pathology. The cell is still the unit or yardstick, but it is not its physical qualities alone which are now

satisfying: its life and habits, its physiology are calling for study. The biology of the cell under normal and pathologic conditions may be accepted as *the* most important scientific problem of today. Many phases of specialized knowledge are necessary to pursue this study, including pathology, chemistry, hematology, physiology and biophysics. This implies cooperative effort. But it may well be expected that as masses of facts are accumulated, another Darwin, with speculative attributes and a judicial mind, will link them into a coordinated web to explain the lawless cell development in the phenomenon known as cancer.

Although it is not primarily a question for the educator, there is one of vital importance to the young man—how to meet the financial burden of the nine or ten years from school through his internship; years when the self-respecting man wants to be self-supporting and in most instances must be self-supporting. At the age of 27 he finds himself theoretically qualified to earn a living in surgery. In reality he cannot expect an appreciable income from private patients much before the age of 40. He wants to know how he can live during this period—which, however, must be a period of preparation, not of waiting. During it, nice judgment is essential to make the minutes count. During this period, character, breadth of mind, knowledge of life are more important than at any other time, and are dependent largely upon his early training which we have recommended should be broadly planned for these attributes. This financial obstacle naturally limits the field materially, and it is found that more and more promising candidates drop out, either from necessity or lack of staying powers. Salaried jobs, governmental positions, private practice, personal indulgences, even an independent personal income, deflect them from the single minded pursuit of a surgical career, which means constant sacrifice, incessant work, and intelligent selection of the lines of effort.

As a matter of history, and years of observation, it is evident that the man *can* find some means of existing, for it was merely that to most of us during these lean years. No single method can be devised to meet all cases, nor can a categorical answer be given to the inquiring and disquieted young man. But he may be assured that his labors and patience will ultimately be rewarded by at least a living wage, and deep satisfaction in the attainment of some success in the career of his choice. Unfortunately, opportunities as private assistants, which formerly kept one going, are now rare, but many full time positions are available in the schools. The privilege of occupying such a position is of inestimable value; the responsibility is great upon him who makes these appointments. Fellowships should be encouraged, and efforts should be directed towards obtaining endowments for this purpose rather than for elaborate buildings and equipment.

Conditions are so rapidly changing in all phases of life that future developments are impossible to foresee. The relation of the profession to the public, as it affects medical practice, is fraught with uncertainty. Industrial and compensation surgery and group medicine are among the factors which are already affecting practice. But the situation in regard to surgery is

fairly clear. That pecuniary returns from private practice will diminish progressively seems certain; the public cannot and will not pay the fancy figures of the past. Moreover, the profession itself is beginning to frown upon the debasement of its reputation and dignity by profiteering. Consequently, commercialism, while not wiped out, will be far less of a temptation than in the past. Idealism and altruism will become more and more the dominating incentives. The high salaries which are paid in some schools to full time clinical workers are sure to be brought more to the plane of the salaries paid to similar grades in the nonclinical departments. The universities are feeling the pinch of the times and this will be one of the first means of curtailing expenses. Further, the 67 four-year schools are now quite adequately supplied with highly trained and competent men in charge of their surgical departments; therefore, there is not the same need as at the early part of the century for training a considerable number of men for academic lives. While each university must develop some younger teachers, only a very few of these will have opportunities for academic careers. The schools must, however, turn out a considerable number of capable surgeons to meet the needs of the community.

Certainly the weakest link in the chain of medical education is in postgraduate surgery. It is well nigh impossible under present conditions to provide operative instruction. A man cannot perform passably or safely as an operator without considerable experience. Theoretical postgraduate courses which include viewing operations or even assisting at operations give a man a dangerous self-confidence and false sense of security. We must face the fact that these men have the right to do surgery and are determined to do it. We must, therefore, accept the obligation of making them safe; in other words, training must be provided. The great teaching institutions are inadequate for this purpose. There are, however, vast facilities in the numerous nonacademic hospitals throughout the country. These should be organized and used for the purpose. The present form of internship is wasteful and should be modified so that the material is shared between interns and other graduate students. The latter should be signed up for a relatively long period and be allowed to operate under instruction and supervision, emphasis being placed as much upon principles as upon technic. The result of their efforts should weigh in the decision as to their qualifications as surgeons.

As urged by our last president, efforts should be more active to control the character of surgery and to protect the public from the incompetent and casual operator. Of course, this applies chiefly in the urban and other thickly populated districts, for in certain rural districts the general practitioner *must* do everything. The license to practice medicine should not include the privilege of doing surgery. In medicine, most conditions being self-limited, a poor practitioner is rarely a serious menace; but give him a scalpel with the patient under an anesthetic and he is dangerous beyond measure.

A man before embarking on surgery should have something more than a medical degree. A certification or registration based upon training, character and ability should be required. The qualifying and examining of a

candidate should be the function of the profession. When the profession has established a standard, its enforcement might well be in the province of the State. We may feel encouraged by the fact that progress in this direction has already been made. An American Board of Surgery is planned to carry out these principles. It should do much to correct this evil and should therefore receive our unqualified support.

The young men for three to five years after their internship might well act as apprentices. This would cut in on the work of the intern staff and attendings, but would do much if adopted by a large number of independent hospitals to meet the minimum requirements to practice surgery. In other words, three years devoted to assisting, operating under supervision, diagnosis, anteoperative and postoperative training should be required to obtain a surgical certificate. Only with such a certificate should a man be qualified to perform major surgery. Our system is antiquated and has not met modern developments. Other countries, led by Denmark, have adopted regulations for recognition as a specialist. They have recognized the indications and have met them by appropriate laws, which it would be well for us to study.

The history of surgery, as we know it, did not exist until the knowledge of bacterial influences and the development of anesthesia quickly changed a limited mechanical field into a vast science. Thereafter, for many years operative technic and the evolution of new and simpler operations constituted the teaching of surgery.

With the relative standardization of technic and of routine operative procedures, this phase is quickly picked up or taught. Diagnosis, judgment and postoperative management cannot be taught, they must be acquired. Original work must be inspired. But example and encouragement must be alive and ever alert to recognize and nurture the rare youth with the essential qualities. Teaching surgery is really a misnomer. Although the surgeon is made and not begotten, and the making in the advanced stages is dependent upon himself, is subjective rather than objective—yet opportunity, help and encouragement are factors which count.

As this is not a lecture to the young man nor a sermon to my peers, I shall not dwell upon the personal attributes such as character, industry, kindliness and tact, which contribute so much to the individual's success. We may accept as the impelling motive the thought phrased by Phillips Brooks, "No man has come to true greatness who has not felt in some degree that his life belongs to his race and that what God gives him he gives him for mankind."

Membership in societies such as this should be, as it is, the reward and recognition of worthy work, appreciable accomplishments, honesty of purpose, and unswerving efforts to uphold the best traditions of the profession.

Anticipation of the forthcoming elaboration of my theme calls to my mind the words of Milton, "I shall detain you no longer, but straight conduct ye to a hillside, where I will point ye out the right path of a virtuous noble education; laborious indeed at the first ascent, but else so smooth, so green, so full of goodly prospect and melodious sounds on every side that the harp of Orpheus was not more charming."

REPORT OF THE COMMITTEE TO STUDY FURTHER PROBLEMS OF POSTGRADUATE SURGICAL EDUCATION IN GENERAL AND THE QUALIFICATIONS FOR SPECIALIZATION IN GENERAL SURGERY IN PARTICULAR *

Evarts A. Graham, M.D.
St. Louis, Mo.

Following the splendid series of papers on the training of the surgeon which were presented at the last meeting, your President, Doctor Archibald, appointed a committee to take such steps as seemed necessary to organize increased facilities for the training of surgeons and to arrange for the certification to the public of properly qualified surgeons. The committee consisted of Drs. Edward W. Archibald, Arthur W. Elting, Evarts A. Graham, Thomas M. Joyce, Thomas G. Orr, and Allen O. Whipple. After some preliminary consideration and correspondence among the members of the committee it seemed probable that it would be necessary to organize a qualifying board of some sort to take care of the certification of surgeons to the public. It also seemed advisable to obtain the coöperation of other surgical societies in this movement. Shortly after the appointment of the committee of the American Surgical Association a committee was appointed by the Surgical Section of the American Medical Association to consider the same matters that were the business of your committee. The group appointed by the American Medical Association consisted of Drs. Fred W. Rankin, *Chairman,* Henry W. Cave, Brien T. King, Roy D. McClure, J. Stewart Rodman, and Hugh H. Trout. Your chairman discussed the matter with the chairman of the Executive Committee of the American College of Surgeons, Dr. George W. Crile, in an effort to enlist the coöperation of the College of Surgeons. As a result of this conversation a meeting was held in Chicago on October 23, 1935, at the building of the American College of Surgeons, at which were present the committees representing the American Surgical Association and the Surgical Section of the American Medical Association, together with representatives of the American College of Surgeons as follows: Drs. George Crile, J. Bentley Squier, Samuel C. Harvey, Irvin Abell, and B. C. Crowell. At a meeting of the American Surgical Association Committee held in the morning of that day at the Stevens Hotel, Chicago, and preceding the meeting with the other committees, it was decided that very probably it would be found necessary to

* Committee appointed by the American Surgical Association at Boston, Mass., June 8, 1935. Evarts A. Graham, M.D., St. Louis, Mo., Chairman.

organize a qualifying board which should be national in scope. As a means of getting action started the following resolution was adopted by your committee and at the afternoon meeting it was presented to the American Medical Association Committee and the representatives of the College of Surgeons:

> A committee appointed by the American Surgical Association to consider the elevation of standards of the practice of surgery and to increase the hospital facilities for the training of young surgeons, proposes that a joint national committee representing the various surgical organizations of the country be formed to discuss and arrange a program for accomplishing those objects. The committee proposes that a national committee of 24 members be formed for this purpose, consisting of six members representing the American College of Surgeons, six the American Medical Association, six the American Surgical Association, two the Western Surgical Association, two the Southern Surgical Association, and two the Pacific Coast Surgical Association. It is hoped that the American College of Surgeons will cooperate in the proposed endeavor by appointing six members to serve on the national committee to organize this work.

After rather prolonged discussion the representatives of the American College of Surgeons agreed to coöperate in this movement, at least to the extent of appointing six members to serve on the proposed National Committee of 24, to discuss ways and means of creating a suitable organization to take care of the qualification of surgeons.

The representatives chosen by the American College of Surgeons to serve on the National Committee were Drs. Samuel C. Harvey, *Chairman,* Irvin Abell, Donald Guthrie, Allen B. Kanavel, C. Jeff. Miller, and Alexander Munroe.

A meeting of the large committee of 24 members was held in Chicago on February 15 and 16 at the Palmer House at which the following were present: Drs. Thomas G. Orr, Allen O. Whipple, and Evarts A. Graham representing the American Surgical Association; Irvin Abell, Samuel C. Harvey, and Alexander Munroe, of the American College of Surgeons; Brien T. King, Roy D. McClure, Fred W. Rankin, J. Stewart Rodman, and Hugh H. Trout, of the Surgical Section of the American Medical Association; Reginald Jackson and Erwin Schmidt, of the Western Surgical Association; and Robert L. Payne and Mont R. Reid, of the Southern Surgical Association. The two representatives from the Pacific Coast Surgical Association could not be present. The New England Surgical Society was not invited to send representatives to the meeting of the National Committee because it would have been necessary to wait for nearly a year for that Society to select its representatives.

The committee organized itself by the election of officers as follows: Drs. Evarts A. Graham, *Chairman;* Samuel C. Harvey, *Vice-Chairman;* and J. Stewart Rodman, *Secretary.* After some preliminary discussion the chairman appointed two subcommittees to meet separately and submit reports before the joint committee on the morning of February 16 on (1) a plan for the organization of an American Board of Surgery, and (2) a plan for increasing the opportunity for the training of surgeons. Dr. J. Stewart Rodman was

appointed chairman of the subcommittee for proposing a plan of organization of the American Board of Surgery. Others of those present who were appointed to serve with him on that subcommittee were Doctors Abell and Munroe, of the American College of Surgeons; King and Rankin, of the American Medical Association; Schmidt, of the Western Surgical Association, and Reid, of the Southern Surgical Association. The chairman of your committee also worked with that subcommittee. Dr. Samuel C. Harvey was appointed chairman of the subcommittee to consider ways and means of increasing facilities for the training of surgeons. Those who served with him on that committee were Doctors Whipple, of the American Surgical Association; Guthrie, of the American College of Surgeons; McClure and Trout, of the American Medical Association; Jackson, of the Western Surgical Association; and Payne, of the Southern Surgical Association.

The reports of the two subcommittees were submitted to the joint committee on February 16 and they were unanimously accepted. The chairman and secretary of the National Committee were authorized to present the plan for the creation of an American Board of Surgery to the Executive Committee of the Advisory Board for Medical Specialties which was scheduled to meet that evening at the Palmer House. The plan was presented to that body and discussed by the chairman and secretary of the committee as authorized and also by Dr. Allen O. Whipple. It was approved by that body.

American Board of Surgery

The details of the plan for the organization of the proposed American Board of Surgery will be worked out by the Board itself. In making the present report we shall limit ourselves to a brief presentation only of the general plan.

It is perhaps not generally appreciated that there are already nine boards representing different medical specialties actively functioning. These boards are respectively for ophthalmology, otolaryngology, obstetrics and gynecology, dermatology and syphilology, pediatrics, psychiatry and neurology, radiology, orthopedic surgery, and finally urology. Recently to this board has been added the newly created American Board of Internal Medicine. All of these various boards have joined with certain national organizations to create the Advisory Board for Medical Specialties which functions in conjunction with the Council of Medical Education and Hospitals of the American Medical Association. The national organizations which have joined with the special boards to form the Advisory Board are the Association of American Medical Colleges, the American Hospital Association, the Federation of State Medical Boards of the United States and the National Board of Medical Examiners. In developing the plan for the proposed American Board of Surgery the scheme of organization of some of the other specialty boards has been followed in the main. In one respect, however, the proposal for the creation of an American Board of Surgery differs from the plan of organization of some

of the other boards in that the responsibility for the American Board of Surgery will rest with the large national and regional surgical associations. For example, it is proposed that the board shall consist of 13 members allocated as follows:

The American Surgical Association	3
The Surgical Section of the American Medical Association	3
The American College of Surgeons	3
The Southern Surgical Association	1
The Western Surgical Association	1
The Pacific Coast Surgical Association	1
The New England Surgical Society	1
	13

It is proposed that each member of the Board shall serve for six years except that lots shall be drawn by the original members of the Board to select certain ones to serve for six years, others for four years and still others for two years in order that a continuity of membership may be preserved so that at the end of six years an entirely new board will not take the place of the old one. It is proposed that a group of Founders be selected to be qualified without examination and that those eligible for the Founders Group will be those holding, when this board begins to function, active and senior membership in the American Surgical Association, the Southern Surgical Association, the Western Surgical Association, the Pacific Coast Surgical Association, and the New England Surgical Society; also professors of surgery, clinical, associate and assistant professors of surgery in all Class A medical schools of the United States and Canada; in addition within the first two years after the Board begins to function, those who have limited their practice to surgery for 15 years may make direct application to the Board and with its approval be certified without examination.

Other candidates who will be qualified by the Board will be expected to present themselves for examination after having fulfilled certain requirements, one of which is that which concerns special training. In the proposed plan the special training shall consist of a period of graduate study of not less than three years beyond the intern year, if taken in a recognized graduate school of medicine, or in an hospital or under a sponsorship accredited by the American Board of Surgery for the training of surgeons. This period of specialized preparation should include sufficient graduate training in anatomy, physiology, pathology, and the other basic medical sciences as are essential to the proper understanding of the practice of surgery, as well as adequate operative experience, evidence of which may be required by the Board. An additional period of not less than two years of study or practice in surgery is required as well.

It is important to note the fact that in the specialized period of training the committee feels that there should be an adequate operative experience.

It would seem far more reasonable to have the surgical neophyte begin his operative experience in an hospital where he will be under supervision than to turn him loose on an unsuspecting public, after an inadequate period of preparation, to acquire his operative experience without supervision which would result, in many instances, in unnecessary deaths and in increased morbidity.

It is proposed that the examinations be divided into two parts of which one will be written and the other practical, covering bedside work, clinical, and operative features. It is also proposed by the committee to have the examinations organized in such a way that part of the practical examination can be devoted to special fields of surgery such as neuro, thoracic, and plastic surgery in order that properly selected candidates may be awarded certificates of qualification in those special fields after they have presented evidence of a satisfactory training and experience in the general field of surgery.

It is proposed that the fee to be paid by members of the Founders Group shall be $25.00 and that a total fee of $75.00 be asked of those who propose to qualify themselves by examination. After the payment of the fees there will be no further dues of any kind. Certificates of qualification by the American Board of Surgery will be issued to the members of both the Founders Group and of the group qualified by examination, after the completion of the payment of the required fees.

Many of the details of conducting the business of the Board will necessarily be determined after it has been organized and after it has created its constitution and by-laws. It would seem wise at this time not to enter into too much discussion of details because they can be better settled by a small group.

A fear has been expressed by some that such a board as planned might attempt to create more standardization of the teaching and practice of surgery than would be desirable. Certainly a too sharply standardized plan of education would be unfortunate. On the other hand, the very method of organization of the board would seem to protect it from the possibility of too much continuous influence in any direction since the members will serve for only six years and will be answerable to their representative organizations which have appointed them. If the Board is not functioning in a proper manner it will be easy enough in the space of two or four years to change its composition to such an extent that a dangerous policy will no longer be continued.

Increasing the Facilities for the Training of Surgeons

It has already been stated that a special subcommittee of the National Committee was appointed under the chairmanship of Dr. Samuel C. Harvey to consider ways and means of enlarging the present facilities for the training of surgeons. In the report of that special committee, which was unanimously adopted by the National Committee, the following recommendations were made.

The American College of Surgeons and the American Medical Association should be asked to form a joint council to organize and carry out a program for the training of surgeons in properly qualified hospitals, setting up such

standards as will meet the requirements of the proposed American Board of Surgery. The standards for the proper training of surgeons shall be subject to the approval of the American Board of Surgery.

The committee also expressed the opinion that in order to provide an adequate number of properly trained surgeons, certain hospitals not now associated with teaching institutions must be developed to the point of carrying out such training. It is hoped that the various hospital associations now organized will give their hearty support to this movement. The committee also emphasizes the fact that a proper graduate training for surgery involves a carefully directed and supervised apprenticeship during which the student should actively participate in practical surgical work, including the performance by himself of operative procedures. He must also have the opportunity at the same time to increase his basic knowledge of the fundamental medical sciences and he must have a thorough and comprehensive training in surgical pathology.

In order to accomplish such purposes the committee feels that hospitals which are selected as being suitable for the proper training of young surgeons should have a type of organization and the facilities which would permit the giving of the training sought. For example, such a hospital must have a continuous ward service of non-private patients under the control of a director, or the equivalent of this, who is responsible for carrying out the program of training of surgeons. This director and his staff must be competent and interested in teaching and willing and able to devote the necessary amount of time to this. It is essential that the relationship between the student and the teacher be a constant and intimate one so that the work of the former is under constant supervision and guidance. It is essential also that such hospitals should have adequate outpatient clinics including follow up and "combined clinics" of which the Tumor Clinic as outlined by the American College of Surgeons is an example. The equipment and facilities of such hospitals must be of proven standards and include adequate laboratories in radiology, pathology, bacteriology, and similar facilities for diagnosis. There must also be an adequate record system and a proper library easily available to the student. Periodic conferences are essential, particularly in relation to the current surgical pathology, the diagnostic problems and the unfavorable results of therapy. Of the greatest importance is the obtaining of autopsies and much emphasis must be laid on the proper ratio between these and the mortality.

It will probably be necessary to have careful and competent periodic surveys of the hospitals designated as suitable for the training of surgeons in order to ascertain that the training is being properly accomplished. Such surveys can be carried out only by surgeons who themselves have been qualified by the Board, are of mature judgment and command the respect of those undertaking to train young surgeons. It is probable also that the progress of the student and the effectiveness of the instruction should be checked by periodic reports to the Council from both the director who is responsible for the teaching in the individual hospital and the men who are students under him.

Recommendations to the American Surgical Association

If the American Surgical Association approves of the report of its committee your committee would suggest that formal approval of the two general suggestions contained in the report be made by the Association. These are, first, the approval of the plan for the creation of an American Board of Surgery and, second, the approval of the suggestion that a recommendation be made to the American College of Surgeons and the American Medical Association that a joint council be organized for increasing the facilities for the adequate training of young surgeons. Again if the Association expresses its approval, the Council of the Association can then consider the matter of the appointment of three members to serve on the proposed board.

As a matter of information your committee is pleased to announce that the Pacific Coast Surgical Association and the executive committee of the New England Surgical Society have already gone on record as approving in principle the recommendations which the National Committee has made. They are the first organizations which have had an opportunity to act upon this important matter. The American Surgical Association will be the third one to have the opportunity to approve of the proposed plan. Your committee hopes that this Association, in which this movement was initiated, will express its approval of the general plans proposed.

Edward W. Archibald,
Arthur W. Elting,
Thomas M. Joyce,
Thomas G. Orr,
Allen O. Whipple,
Evarts A. Graham, *Chairman.*

STUDIES IN BRAIN INJURY
INCREASED CEREBROSPINAL FLUID PRESSURE FROM BLOOD IN THE CEREBROSPINAL FLUID

AN EXPERIMENTAL STUDY

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EXACT knowledge is lacking of the anatomic and physiologic effects of head injury. Experimental study of the problem has been inadequate. Surgeons have carried over into the clinical field of cerebral trauma conclusions reached in physiologic experiments upon the normal animal, with little or no acknowledgment of the fact that the accepted phenomena might not occur in a damaged brain. These propositions were formulated and discussed in a recent review of the literature.[1]

The present studies were undertaken in an attempt to fill in some of the blank spaces of the problem. They have resulted in new data on the relationship of blood in the cerebrospinal fluid to the pressure of the cerebrospinal fluid.

It has been recognized that blood in the cerebrospinal fluid exerts a deleterious influence. Bagley[2] demonstrated neurologic manifestations in dogs after the introduction of blood into the subarachnoid space. An inflammatory reaction in the meninges surrounding the red cells was seen. Essick[3] observed "sterile meningitis" following injection of laked red cells. Bagley[2] recorded dilatation of the ventricle following the introduction of blood, and Wortis and McCulloch[4] and Bagley[2] have shown late fibrosis in the meninges. Many clinical observers[5] believe that blood in the cerebrospinal fluid is harmful. The inflammation caused by the red cells is presumed to hinder the escape of cerebrospinal fluid and hence to raise cerebrospinal fluid pressure.

In 1928 Howe[6] suggested that the blood proteins in a bloody cerebrospinal fluid might be responsible for increased cerebrospinal fluid pressure through osmotic effect. He states: "A similar condition" (*i.e.*, an increase of cerebrospinal fluid through increased osmotic pressure from increased protein content) "occurs if the osmotic pressure of the cerebrospinal fluid is raised by a haemorrhage in the subarachnoid space or when an injection of serum is made into the subarachnoid space." Weed,[7] in 1935 observed that the absorption of cerebrospinal fluid under a constant pressure is retarded by the presence of protein in the fluid. This phenomenon occurred with gelatin or the serum of the animal under experimentation.

METHODS.—Two types of experiments were undertaken. The first consisted of the study of cerebrospinal fluid pressure and anatomic changes following a standard laceration of the brain. On account of a definite association between the pressure curves and the amount of subarachnoid bleeding

observed in these experiments, an attempt was made to separate a rise in pressure due to the increased volume of blood in the cerebrospinal fluid from a rise due to some other mechanism. The general principle therefore of the second type of experiment was the replacement of measured quantities of cerebrospinal fluid by equal quantities of blood and its separate constituents.

Laboratory dogs were employed without choice of breed or sex. In general, large animals were used, the smallest weighing 9.8 Kg. A total of 121 dogs was employed. Food and water were withheld from the animals during the night preceding experimentation.

The anesthetic used in the first group of experiments was intratracheal ether, and in the second intraperitoneal sodium ethyl barbiturate (sodium amytal). The initial dosage of the latter was 50 mg. per Kg. body weight supplemented by additional doses of 5 mg. per Kg. when indicated by beginning restlessness. Control observations indicated that this anesthesia did not affect cerebrospinal fluid pressure, confirming the experience of Milles and Hurwitz.[8]

Physiologic Observations.—Cerebrospinal fluid pressure was observed by means of a straight manometer of 1 Mm. bore, which was filled with physiologic saline. A No. 17 lumbar puncture needle was introduced into the cisterna magna under aseptic precautions. This was connected with the manometer by a glass adapter and about 12 cm. of rubber tubing. During connection of the needle with the manometer, one to two drops of cerebrospinal fluid were lost in avoiding the entrapment of air in the system. Observations of the cerebrospinal fluid pressure at minute intervals were then made for a sufficiently long period to be sure that the pressure was constant within a range of about 15 Mm. This control period was never shorter than 15 minutes in the first group of experiments and 30 minutes in the second group. During the prolonged period of observation after the completion of the experimental procedures, manometer readings were continued at one minute intervals for three hours and at five minute intervals thereafter. The femoral artery was cannulated and a continuous kymographic record obtained of the arterial blood pressure throughout the experiments. The rectal temperature of each animal was recorded at intervals and the normal level of body temperature was maintained with electric pads.

Standard Laceration.—After the control period, a trephine opening was made over the parietal region and a button of bone was removed without injury to the dura. A fitted glass window[9] was immediately screwed into the defect and time was given for the cerebrospinal fluid pressure again to become stable. The window was removed and a sharpened, bent wire was introduced through the dura into the cortex in an area free from cortical vessels and rotated within the substance of the brain from three to five times through 360 degrees. The same wire was used in all experiments, so that the lacerations produced were as nearly uniform as possible. There was no appreciable loss of cerebrospinal fluid during the introduction of the wire. Immediately on withdrawal of the wire the window was screwed back in place, so that the loss of fluid rarely exceeded one drop. Following the laceration the animals were kept under observation for from one to four hours.

Replacement Experiments.—Following the control period, a bulldog clamp was applied to the rubber tubing of the manometer connection about 1 cm. distal to the adapter. A hypodermic needle was introduced into the lumen of the tubing, and 2.0 cc. of cerebrospinal fluid were withdrawn. A second hypodermic needle was then introduced, and 1.5 cc. of the substance to be studied were injected. In order to wash out the system, this was immediately followed by the reintroduction of 0.5 cc. of the cerebrospinal fluid previously withdrawn. In the experiments in which 3.0 cc. of serum were

injected, 3.5 cc. of cerebrospinal fluid were first withdrawn and again 0.5 cc. reintroduced as the final step. In all instances both withdrawal and injection were done at the measured rate of 0.5 cc. per minute. This transient diminution of cerebrospinal fluid volume, with both withdrawal and replacement effected at a slow rate, did not materially affect the cerebrospinal fluid pressure (Chart 5). Pressure readings were begun by removal of the bulldog clamp immediately after completion of the injections.

Control Observations.—(1) Observation of the physiologic set-up without withdrawal or introduction of fluid.

(2) Withdrawal of 1.5 cc. of cerebrospinal fluid and immediate reintroduction of the same fluid.

(3) Withdrawal of 2.0 cc. of cerebrospinal fluid and introduction of 1.5 cc. of physiologic saline solution followed by 0.5 cc. of the cerebrospinal fluid withdrawn.

Experimental Substances Employed.—The appended modifications of blood were used to replace cerebrospinal fluid. In each instance, blood or its fraction was obtained from the dog under observation:

(1) Blood defibrinated by agitation with glass beads.

(2) A suspension of washed red cells. Whole blood was defibrinated and centrifuged. The red cells were washed three times and suspended in physiologic saline, made up to a volume equal to that of the blood treated. Hemolysis was minimal.

(3) A preparation of hemolyzed red cells. After separation of the red cells from defibrinated whole blood, distilled water was added to the former in volume equal to that of the original blood. If laking was not complete, ether was added and later evaporated until the original volume again resulted. 1.5 cc. of this solution, representing the hemoglobin and red cell stroma of 1.5 cc. of blood, were injected. No attempt was made to render the mixture isotonic.

(4) Blood serum. The blood was defibrinated and centrifuged for 15 minutes. A slight degree of hemolysis was present.

All the above materials were prepared and the injections were completed under aseptic precautions.

The animals were kept under observation for a period of from three to six hours. In one instance, observation was carried on for ten hours. Several attempts were made to obtain pressures on succeeding days, which for reasons to be detailed are not being reported.

Pathologic Observations.—At the close of the experiment, the carotids were injected with 10 per cent formalin and the brain was then removed with the dura intact except for the basal portion. After hardening, gross examination of the surfaces and cut sections for obvious lesions was made, followed in the experiments of the second group by microscopic examination of multiple sections through the brain substance and the meninges.

Discarded Observations.—All animals with abnormal body temperature were rejected.

On account of accompanying variability in the cerebrospinal fluid pressure, all animals were discarded in which it was difficult to maintain a steady level of anesthesia.

The occurrence of a bloody cerebrospinal fluid following puncture or of poor respiratory oscillations in the manometer tube, or of the necessity for a second puncture due to dislodgment of the cisternal needle necessitated the abandonment of many experiments.

Experiments in which the arterial blood pressure curves showed greater fluctuations than about 12 Mm. above or below the original levels are not

reported, except in an occasional instance when a greater blood pressure change appeared as a terminal phenomenon after hours of observation.

Experimental Results.—*Standard Lacerations.*—The anatomic lesions created are not uniform. They can be classified in two ways, namely, by

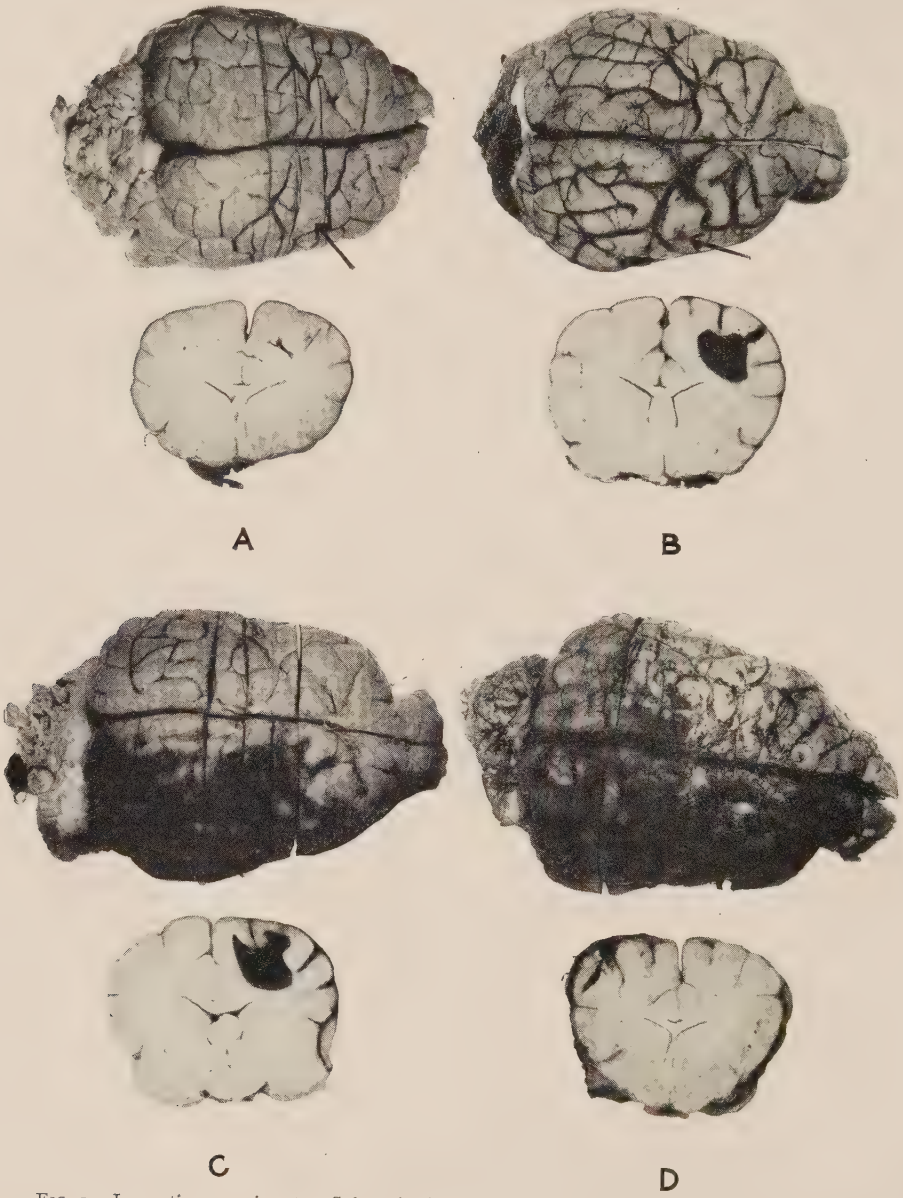

Fig. 1.—Laceration experiments. Subarachnoid bleeding: (A) Grade I. No gross bleeding. Arrow indicates point of entrance of lacerating instrument. Note minimal intracerebral clot. (B) Grade I. Arrow indicates point of entrance of lacerating instrument. Note large intracerebral clot. (C) Grade II. Moderate bleeding. Cross section indicates that the dark area of bleeding in the superior view of the brain is a relatively thin layer of clot. (D) Grade III. Massive bleeding. The superior view of the brain shows wider extent of hemorrhage, and the cross section shows enormously greater thickness of the clot than those shown in Fig. 1C.

the amount of intracerebral clot or by the amount of bleeding into the subarachnoid space.

Comparison between the cerebrospinal fluid pressure curves and the amount of intracerebral bleeding shows no correlation. For instance, there is obviously a considerable increase in volume of the brain shown in Figure 1B as compared with that shown in Figure 1A, and yet the pressure curves (Chart 1) both remain at the control level.

On the other hand, there is a direct correlation between the cerebrospinal fluid pressure and the amount of bleeding into the subarachnoid space. A classification based on the degree of subarachnoid bleeding may therefore be made. No gross bleeding into the subarachnoid space has been classified as Grade I; moderate bleeding as Grade II, and marked bleeding as Grade III.

In Grade I (Figs. 1A and 1B) the cerebrospinal fluid pressure does not vary materially from the original reading (Chart 1).

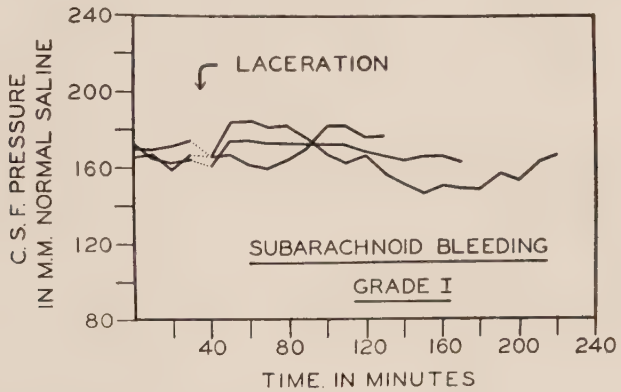

Chart 1.—Laceration experiment. No gross subarachnoid bleeding. The arrow in this chart and in Charts 2 and 3 indicates the time at which the laceration was effected. Note the absence of immediate disturbance of cerebrospinal fluid pressure from the experimental manipulations and the absence of any consistent rise in spite of extensive brain laceration.

In Grade II (Fig. 1C), there is an immediate moderate rise of cerebrospinal fluid pressure, occurring during the first 20 minutes, followed by a sustained elevation slightly lower than the maximum pressure (Chart 2). It will be noted that in one of these experiments the cerebrospinal fluid pressure curve does not rise. No explanation for this atypical observation can be presented.

In Grade III (Fig. 1D), there is an immediate rise of cerebrospinal fluid pressure of enormous extent, occurring in the first five minutes (Chart 3). This high peak is followed by a less abrupt fall, the pressure reaching within two hours a fairly constant level, elevated above the original pressure.

Replacement Experiments.—Control experiments, consisting of (1) prolonged anesthesia in the intact animal, (2) withdrawal and reinjection of cerebrospinal fluid, and (3) replacement of cerebrospinal fluid by physiologic salt solution, demonstrate no significant change in cerebrospinal fluid pressure (Charts 4 and 5).

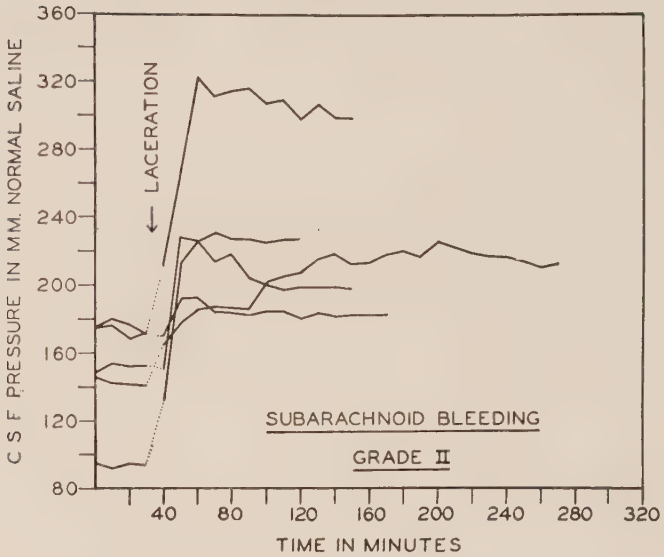

CHART 2.—Laceration experiment. Moderate subarachnoid bleeding. Note immediate rise of cerebrospinal fluid pressure with slight tendency to later fall in most experiments.

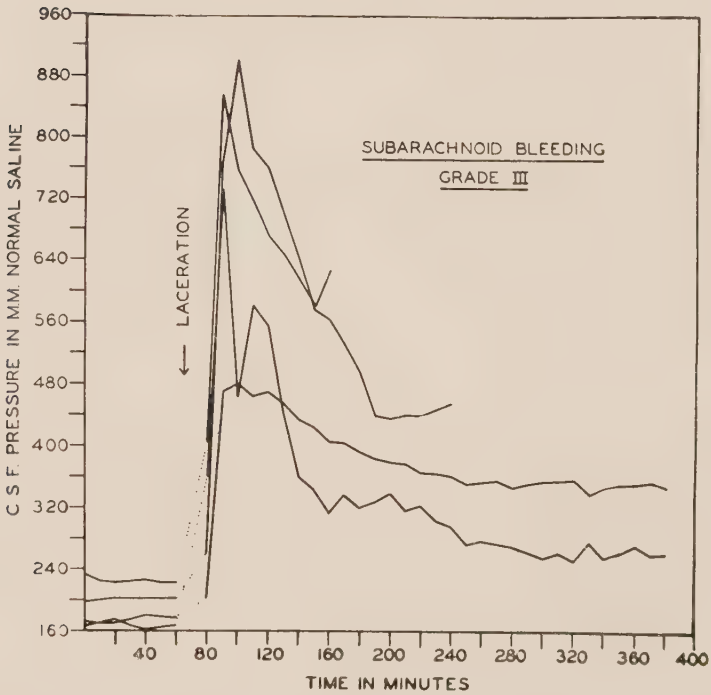

CHART 3.—Laceration experiment. Massive subarachnoid bleeding. Note that the scale of this chart has been halved as compared with Charts 1 and 2. Note also the rapid sharp rise to as much as 900 Mm. of saline followed by a slower drop and a later sustained elevation.

After the replacement of 1.5 cc. of cerebrospinal fluid by the same quantity of defibrinated, whole blood, the cerebrospinal fluid pressure rises steadily (Chart 6). This rise occurs at about the same rate in all experiments except one, in which, after a typical early rise, a slight drop from the maximum pressure occurs.

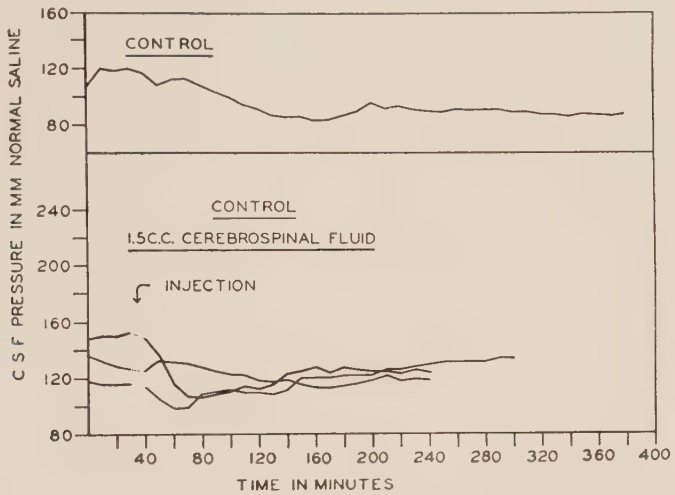

CHART 4.—Replacement experiment control. The upper curve represents the cerebrospinal fluid pressure in the anesthetized animal without other procedure. The lower curves represent withdrawal from and immediate reinjection into the cisterna magna of 1.5 cc. of cerebrospinal fluid. The arrow in this and succeeding charts represents the time at which injection was made.

After the replacement of cerebrospinal fluid by a suspension of washed erythrocytes, obtained from 1.5 cc. of whole blood, there is no rise of cerebro-

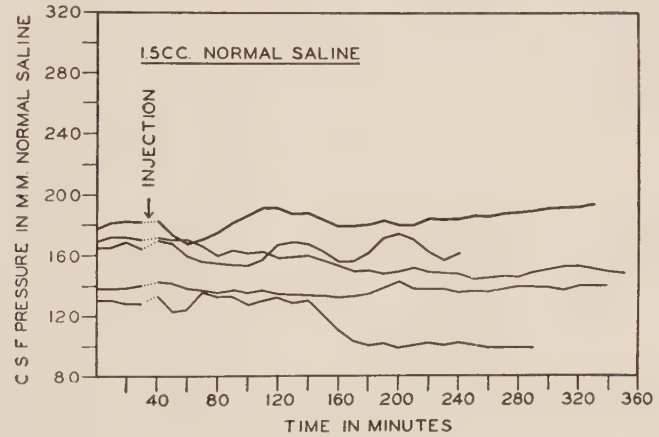

CHART 5.—Replacement experiment control, 1.5 cc. of normal saline.

spinal fluid pressure for as long as five hours (Chart 7). The individual curves differ from those of the control experiments only in slightly greater irregularity of pressures. One curve shows a definite rise in pressure for which no proven explanation can be offered. Accidental bleeding, unde-

tected on account of discoloration already present from the injected red cells, offers a possible solution.

Three experiments were performed with repeated estimations of cerebrospinal fluid pressure at one or two day intervals for as long as five days after the introduction of washed red cells. In no instance was there observed any

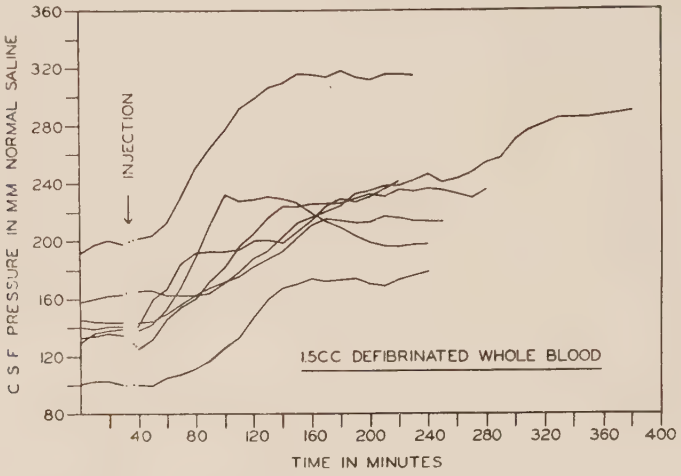

Chart 6.—Replacement experiment, 1.5 cc. defibrinated whole blood. Note consistent rise of cerebrospinal fluid pressure. One curve after a typical initial rise shows a tendency to fall.

increase over the original cerebrospinal fluid pressure. These experiments are not reported in detail because of the possibility of leakage of cerebro-

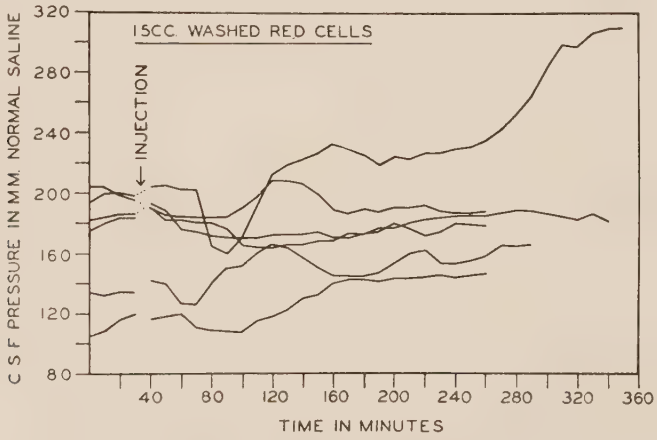

Chart 7.—Replacement experiment, washed red cells from 1.5 cc. of blood. Note somewhat irregular character of curves without consistent tendency towards elevation of pressure. One curve shows a definite rise in cerebrospinal fluid pressure.

spinal fluid from earlier punctures and because of lack of uniformity from day to day in anesthesia and water balance.

After the replacement of cerebrospinal fluid by the hemolyzed red cells from 1.5 cc. of blood, the cerebrospinal fluid pressure rises in contrast to its behavior when the intact cells are introduced (Chart 8).

After the replacement of 1.5 cc. of cerebrospinal fluid by 1.5 cc. of blood serum, the cerebrospinal fluid pressure rises with great regularity (Chart 9). The angle of elevation is somewhat greater than that when whole blood is introduced and markedly greater than that when hemolyzed red cells are introduced. In one experiment carried out for ten hours, no drop in pressure occurred.

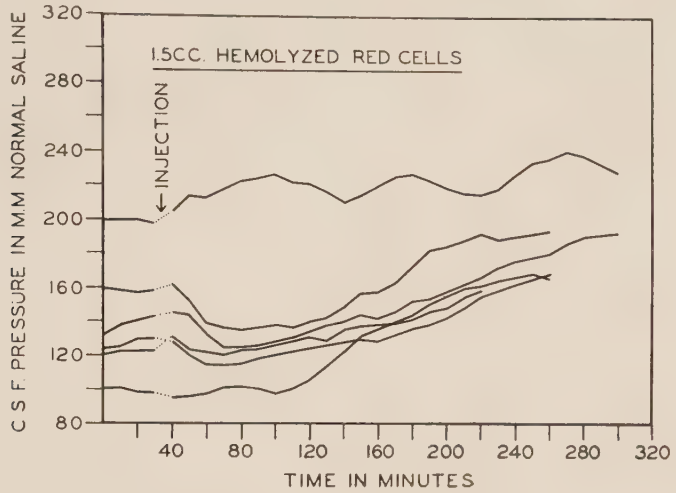

CHART 8.—Replacement experiment, hemolyzed red cells from 1.5 cc. of blood. Note slight elevation of cerebrospinal fluid pressure.

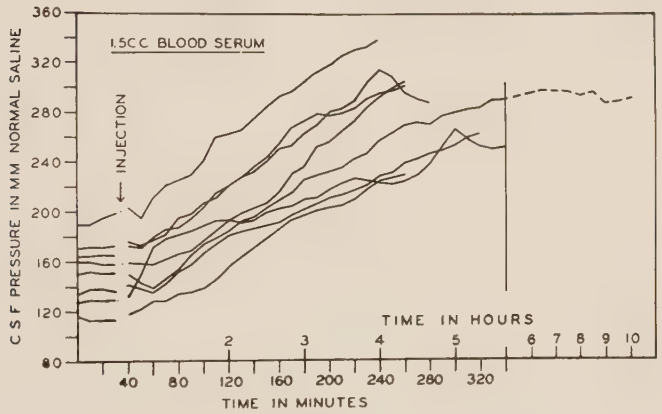

CHART 9.—Replacement experiment, 1.5 cc. of blood serum. Note regularity of rise in cerebrospinal fluid pressure which is greater than in any previous experiment. In one experiment, observation continued over ten hours shows a sustained elevation of pressure without tendency towards further rise or fall.

When the amount of blood serum so introduced is increased to 3.0 cc., the rise of cerebrospinal fluid pressure is markedly increased both in rate and in ultimate elevation (Chart 10).

Comparison of these differences in reaction of the cerebrospinal fluid pressure can best be made by comparing the average curves following the introduction of blood and its constituents (Chart 11).

Histologic Findings.—Pathologic changes in the material studied from the replacement experiments were limited to the meninges. No evidences of

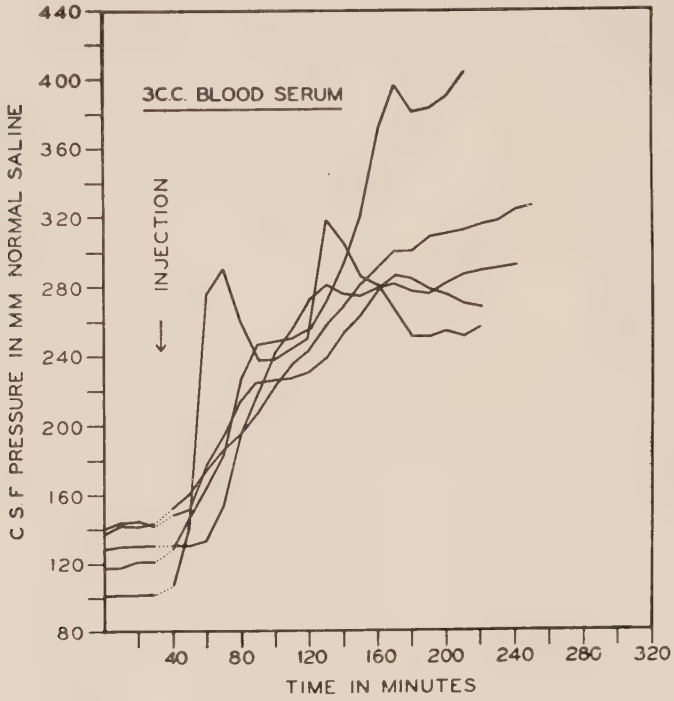

Chart 10.—Replacement experiment, 3.0 cc. of blood serum. Note sharper angle of rise and greater ultimate elevation than those recorded in Chart 9.

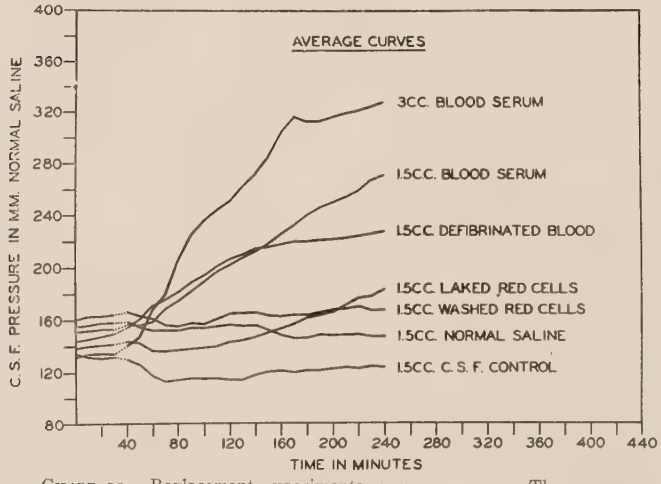

Chart 11.—Replacement experiments, average curves. The curves are constructed by averaging, at ten-minute intervals, the pressure readings from the individual experiments recorded in Charts 4 to 10 inclusive.

trauma, inflammation or edema were found within the substance of the brain in any instance.

The typical meningeal changes observed consisted of infiltration of the arachnoid and of the pia with monocytes, lymphocytes, and occasional polymorphonuclear leukocytes. A striking variability of reaction, both in incidence and distribution, was evident. For instance, five out of six brains studied following the injection of 1.5 cc. of blood serum showed a slight to moderate inflammation, whereas the sixth brain showed almost as intense a reaction as was observed in any experiment. Particularly in those instances of less reaction, the distribution of the areas of inflammatory change was irregular. Large areas of the meninges appeared entirely normal. The areas of pathologic change tended to occur most frequently about the base of the brain near the point of cisternal puncture.

A rough determination of the average intensity of the cellular reaction in each group of controls and experiments was attempted (Table I). It is realized that such averages are only approximate on account of the variations in incidence already cited.

TABLE I

COMPARISON BETWEEN CEREBROSPINAL FLUID PRESSURE RISE AND AVERAGE ESTIMATED INTENSITY OF CELLULAR INFILTRATION OF MENINGES

Exp.	No. Brains	C. S. F. Pressure Rise	Meningeal Inflammation
Control	5	0	+
Washed R. B. C.	6	0	++++
Laked R. B. C.	3	+	++++
Whole Blood	4	++	++++
Serum, 1.5 cc.	6	+++	++
Serum, 3.0 cc.	4	++++	++

DISCUSSION.—The three types of cerebrospinal fluid pressure curves in the three grades of subarachnoid bleeding following experimental laceration of the brain cannot be interpreted with entire satisfaction. The absence of pressure rise in the absence of gross subarachnoid bleeding seems significant as indicating no effective increase of brain volume from intracerebral clot or from possible traumatic edema. The sharp rise when moderate bleeding occurs may be ascribed to the increase of volume due to the effusion of blood, which is unquestionably the cause of the enormous rise when massive bleeding occurs. The succeeding drop in the latter curve probably represents adjustment of intracranial blood volume, and perhaps of cerebrospinal fluid volume, to this additional content of the closed space. It is noted, however, that a sustained elevation is established after this presumed adjustment has taken place. This observation led to the replacement experiments in an attempt to determine whether some added factor besides volume increase may be responsible for increased cerebrospinal fluid pressure in the presence of bleeding.

Such an added factor is suggested by the behavior of the cerebrospinal fluid pressure after the injection of whole blood, hemolyzed red cells and

blood serum. On account of the contradiction offered by the results of these experiments to current surgical opinion, it is important to repeat that the intact red cells are not effective in elevating cerebrospinal fluid pressure during the first few hours.

The definition of the factor common to the three effective materials must be attempted and the mechanism of its effect on cerebrospinal fluid pressure considered. Following the injection of any alien material into the cerebrospinal fluid spaces, under conditions in which the content of the spaces is not immediately altered, the pressure may rise as the result of three fundamental changes, namely, (1) an increase in the volume of the brain from edema, (2) an increase in the blood volume within the cranial cavity, and (3) an increase in the amount of cerebrospinal fluid.

Neither an increase of volume of the brain from edema nor an increased intracranial blood volume seems to be related to the rise of cerebrospinal fluid pressure in these experiments. If either of these phenomena occurs, one would expect it to be expressed in rises of pressure in the control and particularly in the washed red cell experiments. The histologic studies (Table I) suggest that the red cells are among the most irritative of the materials injected. None of the brains presented histologic evidence of edema. An increase in blood volume should not give a sustained elevation of pressure over a period of ten hours (Chart 9), nor should the rise in pressure be so gradual.

It seems logical, therefore, to assume that the phenomena recorded are the result of increase in the amount of cerebrospinal fluid. Under the conditions of these experiments, such an increase could result either from inflammation or from an increase in the osmotic pressure of the fluid.

That inflammation may occur is shown by the present microscopic studies as well as by the work of Essick[3] and Bagley.[2] Table I, however, demonstrates no correlation between the magnitude of rise of cerebrospinal fluid pressure and the degree of inflammatory reaction.

This finding is emphasized by several isolated observations. In the instance already cited from the group of experiments with 1.5 cc. of serum, an inflammatory reaction of great intensity in one animal was accompanied by no more prompt, more rapid or more marked rise in pressure than occurred in the remaining five animals in which the reaction was slight. In a single control in which cerebrospinal fluid was removed and reinjected, the meninges showed an intense purulent meningitis. In this instance no pressure rise occurred. In the animal injected with 3.0 cc. of serum that showed the most marked pressure rise of the entire series, the inflammatory reaction was slight to moderate in degree.

If it is then concluded that, although inflammatory changes occur, they are not causally related to an increase in the volume of cerebrospinal fluid, there remains the factor of osmotic pressure to consider. The differences in amount of cerebrospinal fluid pressure increase with the injection of different substances may be expressed in terms of percentage change (Chart

12). The most striking quantitative relationship thereby demonstrated is the almost exact doubling of the pressure rise with the doubling of the amount of serum injected. The failure of the pressure to rise as high following the injection of 1.5 cc. of defibrinated whole blood, as following the injection of an equal quantity of serum, is explained by the presence of red cells, previously shown to be inert. These quantitative relationships are entirely consistent with the behavior of an osmotic process. The difference in pressure response to the injection of washed red cells and laked red cells is explained by the fact that hemoglobin can exert an osmotic effect in the latter instance and not in the former. Inasmuch as the serum proteins, par-

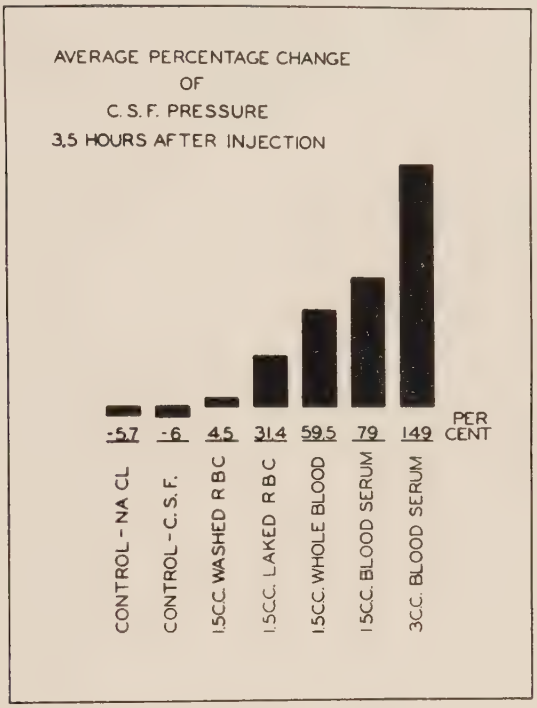

CHART 12.—Replacement experiments. The percentage values here shown were calculated from Chart 11. They represent the average amount of rise or fall of cerebrospinal fluid pressure expressed as a percentage of the average original pressure in each group, at the end of three and one-half hours.

ticularly the serum albumin, are responsible for the maintenance of the normal osmotic tension of the circulating blood, it is highly probable that the introduction of these proteins into the cerebrospinal fluid will upset its normal osmotic balance. This conception agrees with Howe's[6] statement and is confirmed by the recent experiments of Weed.[7]

The clinical application of this study relates to the problem of increased intracranial pressure. Although the clotting elements are not present in the blood introduced experimentally, yet there seems to be no reason to expect

that the serum proteins of blood in the subarachnoid space of the patient would not behave just as they have apparently behaved in these experiments. In other words, in any case with blood in the cerebrospinal fluid, an increase in cerebrospinal fluid pressure may be due to two factors: the increase in volume due to addition of blood and the increase in volume due to osmosis. That the phenomena here reported may have clinical significance is indicated by the fact that the introduction of 3.0 cc. of serum into the dog's cisterna after the withdrawal of 3.0 cc. of cerebrospinal fluid will elevate the cerebrospinal fluid pressure to an average level of 340 Mm. of normal saline.

It is unwise to formulate any practical conclusions in regard to the treatment of hemorrhage into the subarachnoid space. Although apparently logical procedures might be suggested, the unknown factors operative in the individual case are still too many and too complex to permit therapeutic generalizations. Certainly any inclusive program for the treatment of increased intracranial pressure must take into consideration the phenomena here described.

CONCLUSIONS

(1) In experimental laceration of the brain in the dog, the cerebrospinal fluid pressure varies directly with the amount of blood that escapes into the subarachnoid space, and not with the amount of bleeding within the cerebrum.

(2) In spite of the previous withdrawal of an equal quantity of cerebrospinal fluid, a rise in cerebrospinal fluid pressure occurs in the dog following the introduction of the following substances into the cisterna magna: (1) a solution of hemoglobin; (2) defibrinated blood; and (3) blood serum.

(3) The introduction of twice the quantity of blood serum will approximately double the percentage rise of cerebrospinal fluid pressure.

(4) The introduction of washed red cells produces no increase in cerebrospinal fluid pressure over a period of as long as five hours.

(5) Microscopic study of the brains following the partial replacement of cerebrospinal fluid by blood and its separate constituents reveals inflammatory changes, which are not correlated with cerebrospinal fluid pressure changes.

(6) It is probable that the phenomena reported are the result of an increase of osmotic pressure of the cerebrospinal fluid due to the introduction of blood proteins.

(7) The approach to the problem of increased intracranial pressure must include consideration of the phenomena of osmosis as operative in bloody cerebrospinal fluid.

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REACTIONS FOLLOWING OPERATIONS FOR HYPERTHYROIDISM

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AMONG the achievements of modern surgery in the treatment of exophthalmic goiter, perhaps the most tangible, and certainly the most gratifying, has been the remarkable reduction of the mortality. A comparison of the mortality occurring among patients operated upon at The Mayo Clinic for exophthalmic goiter during the last five years of the preiodine period (1918 to 1922, inclusive) with that among patients operated upon during the past 12 years (1924 to 1935, inclusive), shows that while of 3,636 patients who were operated upon in the former period, 119 died, or a mortality of 3.27 per cent; of 9,223 patients who were operated upon in the latter period, only 76 died, or a mortality of 0.82 per cent.

This, however, tells only part of the story, for many more patients with exophthalmic goiter have been saved than is indicated by the difference in the surgical mortality. In the same preiodine period referred to, the percentage of patients who died before they could be prepared for operation (2.06 per cent) nearly equaled the number who died following operation, whereas in the latter period this figure was reduced to 0.34 per cent. In addition, during the preiodine period, when in approximately two-thirds of the cases preliminary ligations of the thyroid vessels were necessary to prepare patients for subtotal thyroidectomy, there was an interim mortality among patients who had returned home following such procedures, which, though it could not be estimated accurately, was not inconsequential. Since, today, the need for preliminary ligation is reduced to a very small percentage of the cases, this interim mortality has been virtually eliminated. It becomes apparent, therefore, that the total death rate for exophthalmic goiter during the preiodine period was at least eight times greater than it is today; or to express it in different terms, today, of every eight patients with exophthalmic goiter who formerly would have died, seven are saved.

Measured, then, by the results attained only a score of years ago, the amazingly improved results of today can be justly viewed with pride and enthusiasm. However, in our rejoicing it is important that we be on guard lest excessive enthusiasm lead us complacently to accept the present results as the final limit of progress. That there are still unsolved problems associated with the treatment of exophthalmic goiter is evident when the surgical mortality of this disease is compared with that of adenomatous goiter without hyperthyroidism (simple goiter), the mortality for the former condition being approximately ten times greater than that for the latter. The cause of this higher mortality among patients with exophthalmic goiter can be ac-

counted for in some instances by the presence of visceral injuries sustained prior to operation as the result of prolonged hyperthyroidism. In other instances in which patients die following operation the cause of death cannot be adequately explained by the anatomic findings at necropsy, and we are forced to ascribe death in such cases to acute hyperthyroid reactions. Although the problems created by these factors are not today as pressing as they formerly were, they should not be lost sight of. Instead they should be kept in the foreground constantly to challenge our best efforts.

In analyzing the records of the 76 patients with exophthalmic goiter who have died at the clinic following operations since the standardization of iodine treatment, it is not always possible to determine the principal cause of death, since in many instances death is not the result of a single factor but of a combination of two or more factors. However, such an analysis does permit of a general classification of the principal surgical hazards into three groups: (1) those connected with the operative procedure; (2) those dependent on the presence of a coexisting or associated disease, and (3) those inherent in the disease of exophthalmic goiter itself.

The first group of hazards includes not only accidents of technic, which are for the most part avoidable, but also errors of surgical judgment, including, in particular, the decision as to when to operate and the selection of the proper surgical procedure. In about 10 per cent of the cases in which death occurred, the operation was complicated by a technical mishap. While these errors were not necessarily of a nature to cause death, they must be considered as contributing factors.

The second group of hazards is encountered in cases in which other diseases are associated with exophthalmic goiter. This factor of associated disease plays a greater rôle in mortality attending operations for exophthalmic goiter than in that attending operations for many other diseases. It is not because the incidence of associated disease is necessarily higher among patients with exophthalmic goiter, but rather it is because operation in these cases, in which exophthalmic goiter is complicated by another disease, is undertaken more often than on patients suffering from other surgical conditions similarly complicated. Rarely does the presence of associated disease constitute a definite contra-indication to operation in cases of exophthalmic goiter; on the contrary, the indications for operation often become more urgent, since relief of the hyperthyroidism is either beneficial to the associated disease or is necessary before its proper treatment can safely be instituted. The presence of an associated disease, including active tuberculosis, brain tumor, angina pectoris, carcinoma of the suprarenal glands with metastasis, pyelonephritis, Addison's disease and Parkinson's disease, constituted the principal factors in 10 per cent of the deaths in this series.

The third group of hazards constitutes a problem far more complicated and less tangible than the others and represents the most important one in the surgical mortality in exophthalmic goiter. While not always independent one of the other, the hazards inherent in the disease itself may be con-

veniently subdivided into two classes: (1) those dependent upon the presence of visceral injuries, and (2) those dependent upon postoperative metabolic reactions. Of the visceral lesions found at necropsy of patients who have died of exophthalmic goiter, the most common and the most thoroughly investigated are those involving the heart and liver. Although cardiac hypertrophy[8] and dilatation are not uncommon findings at necropsy, rarely do patients with exophthalmic goiter die of congestive heart failure. What contributing influence these complications have on the operative mortality cannot be accurately evaluated. Because of their relatively irreparable nature, the hepatic changes are perhaps the more serious.

In a previous study of the pathologic anatomy of the liver, with correlated clinical findings in 107 cases of exophthalmic goiter, Beaver and I[2] found three types of hepatic lesions predominating: (1) acute degeneration (fatty metamorphosis, focal and central necrosis and changes secondary to stasis of blood), (2) simple atrophy, and (3) subacute toxic atrophy and cirrhosis. The lesions were intimately related in severity to the intensity and duration of the hyperthyroidism. Since it was estimated that in approximately 40 per cent of these cases the severity of the lesion was sufficient to impair function, it is logical to conclude that hepatic injury has a significant influence on the mortality in exophthalmic goiter. Of the 53 cases in this series in which necropsy was performed, the pathologist reported gross anatomic changes in the liver or heart, or both, in 26 cases.

The surgical problems involved by these factors are (1) one of prevention, and (2) preoperative recognition and treatment. Since the development of secondary lesions in the liver and heart are largely dependent on the intensity and duration of hyperthyroidism, they can be virtually prevented by the institution of prompt surgical treatment. The importance of the duration of the disease as an influence in the surgical mortality is forcibly emphasized when the average duration of the disease in all cases of exophthalmic goiter in which patients were operated on since 1923 is compared to that in those cases in which the patients died. For the entire series the average duration of the disease was 17.70 months, whereas for the group who died it was 28.09 months.

While the presence of visceral injuries can commonly be detected prior to operation by means of clinical tests of hepatic efficiency, the electrocardiogram, and other clinical means of investigation, their significance on the operative hazard cannot always be accurately determined, since a very high percentage of patients with known visceral injuries easily endure the operation. However, knowledge of the presence of such visceral injuries is important, since it clearly indicates that additional measures of safety may be required in the preparation and treatment. These include, in particular, prolongation of the usual period of preparation and the employment of stage procedures in resection of the goiter.

The clinical response of the patient with exophthalmic goiter to the surgical procedure, which is manifested in part by an intensification of already

accelerated metabolic processes, commonly referred to as the "postoperative reactions of hyperthyroidism," are today much less frequent and severe than they were in the preiodine period. This change is particularly noticeable in the virtual elimination of the typical postoperative crises of the disease, which resemble the picture of an overwhelming intoxication; formerly these crises were frequently precipitated even by minor procedures, such as ligation or injection of a few cubic centimeters of quinine-urea solution into the gland. Commonly the reaction begins within a few hours after completion of the operation and it is manifested by increasingly marked tachycardia, intense flushing and sweating, nausea, persistent vomiting, rapid respirations, progressive mental agitation and restlessness, which causes the patient to thrash about in bed almost incessantly. The temperature rises with the onset of the reaction and within a few hours reaches from 102° to 104° F. or higher. Frequently, delirium, prostration and coma supervene. While occasionally the symptoms abate after two or three days, in most instances death follows in from 12 to 48 hours and at necropsy no anatomic changes can be found to account for it. The picture is almost identical to that of the spontaneous crisis of the disease, with the difference that in the latter the course is less rapidly progressive and is usually unaccompanied by fever, unless complicated by infection, until the terminal stages.

The abnormal physiologic process involved in the precipitation of a reaction is not fully understood. According to the theories which seem the most tenable the mechanism by which the reaction is produced is explained: (1) by the sudden increase in the amount of thyroid secretion (either normal or abnormal) in the tissues, and (2) by hypersecretion of epinephrine. Since increased oxidation in exophthalmic goiter is caused by increased thyroid secretion, and since the clinical manifestation of the reaction can be explained at least in part by an acceleration of the processes of oxidation, it is logical to assume, in the absence of proof to the contrary, that postoperative reactions are caused by a further increase in thyroid secretion. That surgical manipulation of the thyroid gland is not necessary to precipitation of such a reaction is evidenced by the fact that reactions have been produced in cases of exophthalmic goiter by operations performed for conditions other than goiter. However, there are reasons to doubt that the thyroid gland is capable of suddenly discharging sufficient thyroxin to produce such marked postoperative reactions as are sometimes seen. There is evidence to indicate that the intensity of the response to intravenous injection of thyroxin varies inversely with the patient's basal metabolic rate. Thus Thompson and his associates[14] estimated that the intravenous injection of 10 mg. of thyroxin produces about seven times as much effect when given to patients whose basal metabolic rate is —40 per cent as when given to patients with normal levels of metabolism. In 1920 Plummer administered intravenously, to several patients with exophthalmic goiter whose basal metabolic rate was +75 per cent or greater, 15 mg. of thyroxin every second day for four doses.

These injections resulted in no apparent response, either in elevation of the basal metabolic rate or in the development of any clinical effects.

The theory that these reactions are caused by hypersecretion of epinephrine, induced by physical and emotional factors associated with the operation, is supported in part by the following experimental and clinical observations: (1) the functional activity of the suprarenal medulla is increased by pain and other major emotions, as shown by Cannon,[3, 4] (2) injection of adrenin induces secretory activity of the thyroid gland, as shown by Cannon and Cattell,[4] (3) thyroxin renders the sympathetic nervous system more excitable to the action of epinephrine in raising arterial blood pressure, as shown by Levy,[10] (4) the symptoms of hyperthyroidism abate after denervation of the suprarenal glands, according to Crile,[5] and (5) the response of a patient with hyperthyroidism to the subcutaneous injection of epinephrine hydrochloride is similar in nature to the hyperthyroid reaction following operation, as reported by Goetsch and Ritzmann.[6]

While today the typical crisis (both postoperative and spontaneous) is uncommon, there still occurs, perhaps after every operation for active exophthalmic goiter, some reaction of hyperthyroidism. When the patient has been adequately prepared with iodine the reaction is commonly so mild that, clinically, it can scarcely be distinguished from the normal postoperative response of a patient with simple goiter, and it differs only in increased sweating, a moderate rise in temperature and pulse rate, and perhaps in the development of intermittent irregularity of cardiac rhythm. As a rule such symptoms do not develop immediately after operation but are delayed for six to 12 hours or even longer, and they usually subside within from 24 to 48 hours without having materially influenced the patient's convalescence. The more severe hyperthyroid reactions seemingly differ from the milder ones only in the intensification of the symptoms, and usually they are not accompanied by the extreme mental agitation, the restlessness and the general toxic state which characterize the typical crisis of the disease such as formerly was observed among patients who had not received iodine. Unlike the typical crisis of the preiodine period, the intense metabolic reactions as seen today do not usually appear sufficient in themselves to cause death. In some cases, death under these circumstances can be more readily explained on the basis of the failure of the liver, and perhaps other vital organs already impaired, to respond to the additional load imposed by the reaction. In other cases death is not infrequently attributable to infectious processes activated because of lowered resistance of the patient induced by the reaction.

Today, the intense metabolic reactions are for the most part limited to a relatively small group of cases of severe or relatively severe hyperthyroidism in which there is a large firm goiter of long duration. Many children with exophthalmic goiter also fall into this group. Since the condition of the patients in this group cannot be improved materially by any known measure short of partial removal of the goiter, and since the operative mortality among them is relatively high, there is obviously the need of some other

therapeutic measure which will either abate the intensity of the hyperthyroidism or will fortify the patient better to endure the postoperative reaction.

From the field of research into the relationship of the thyroid gland to other organs of internal secretion there have already come sufficient clues to further the hope that a final solution of some of these problems will be found. That the suprarenal cortex has some inhibiting effect on the thyroid gland seems to be well established. Many investigators have shown that enlargement of the suprarenal gland follows administration of thyroid substance. Marine and Baumann[11, 12] and others have suggested that, in exophthalmic goiter, the suprarenal gland exerts an influence which is antagonistic to overactivity of the thyroid gland. They have prepared an extract from the suprarenal gland and employed it in cases of hyperthyroidism, but without significant improvement of the patient.

Kendall[9] has suggested administration of sodium chloride and sodium bicarbonate, or sodium citrate, together with cortin as a means of combating postoperative hyperthyroid reactions. This suggestion is based on the following reasoning: Koelsche[9] has shown that in adrenalectomized animals, active preparations of the cortical hormone exerted a sparing effect on the breakdown of protein. Allers[1] has extended this observation and found that sodium chloride alone lessens the severity of the breakdown of protein after the administration of thyroxin. In addition, Allers has demonstrated that an adrenalectomized animal can be maintained in a normal condition, without cortical hormone, provided sufficient sodium chloride and sodium bicarbonate (or sodium citrate) are given to replace the daily loss of these salts.

This work, which has been confirmed by Harrop and his co-workers,[7] emphasizes the close relation between the action of the cortical hormone and mineral metabolism, particularly sodium salts. In the condition of crisis which is observed in acute cases of exophthalmic goiter, it seems probable that in many cases there is a negative balance of sodium salts, particularly sodium chloride. The administration of sodium chloride and sodium bicarbonate is indicated; but, provided the suprarenal cortex has been stimulated to the point of exhaustion, the administration of sodium salts alone might give only temporary relief. By the injection of sodium chloride and sodium bicarbonate, or sodium citrate, together with a potent extract of the suprarenal cortex, the deficiency in mineral metabolism would be remedied and the possible deficiency in cortical hormone relieved.

Since mild and moderately severe reactions commonly subside spontaneously within from 24 to 48 hours, the application of this form of treatment has been limited to four cases in which postoperative reactions of severe intensity developed and the patients were considered to be critically ill. Of these four patients, three had been operated upon for exophthalmic goiter, the remaining one for adenomatous goiter with hyperthyroidism. In two of the cases of exophthalmic goiter marked clinical improvement apparently resulted from the intravenous administration of a solution of 5 per cent sodium citrate and 0.9 per cent sodium chloride, to which was added an active prep-

aration of cortin. One patient, a girl, aged 12, received two injections of 850 cc. each of this salt solution, on successive days, containing 5 cc. and 10 cc., respectively, of cortin. The other, an adult, received two injections, eight hours apart, of 500 cc. of the salt solution containing 15 cc. of cortin. Both patients made a satisfactory recovery. Unfortunately in the first case estimations of the chemical constituents of the blood were not undertaken. In the second case the values for blood potassium, chlorides and sodium were determined before operation and at frequent intervals after operation. The value for blood potassium on the second morning after operation was slightly elevated, but it returned to normal following the intravenous injection. The values for blood chlorides and sodium remained normal throughout the period of observation.

It is apparent that no conclusions regarding the value of administering sodium chloride and sodium citrate together with a potent extract of suprarenal cortex are warranted from this very limited experience, and this form of treatment is mentioned here only in the hope that from this or from other fields of investigation there may be developed a treatment which will prove of value in preventing or combating the postoperative reactions of hyperthyroidism which today represent a most important factor in the surgical mortality in a small group of patients with exophthalmic goiter who cannot be made safe operative risks by measures now available.

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DISCUSSION.—DR. FRANK H. LAHEY (Boston, Mass.).—It seems to me that thyroid deaths are largely liver deaths. There are many factors which indicate this. Another thing is that some thyroid deaths each year appear to be inevitable and unavoidable.

We do about 1,000 goiters a year; we have performed some 14,600 operations upon approximately 12,000 people. Certainly one should be able to avoid mortality, but we do not seem to have been able to do it. Doctor Pemberton and Doctor Crile have probably performed more operations, and many of you have done as many, but we all still have mortalities with hyperthyroidism. It, therefore, becomes extremely important, I think, for us to concentrate on what are the factors that are indicative of the severity of hyperthyroidism and which may predispose to a fatality.

The reason, I think, that some of these patients die liver deaths is because those who die with no operation are frequently jaundiced; they die with temperatures of 105° to 106° F.; they are benefited by the things that benefit diminished liver function, particularly fluids and glucose, and the disease is really a disease not of intoxication but of excessive combustion.

Some of the factors which are helpful in selecting risks, and thus in selecting graded operations, are of extreme importance. I would strongly urge that pole ligation should not be given up. I think that because we have come to lean on iodine, we tend to believe that we do not need pole ligation. There will shortly appear in Surgery, Gynecology and Obstetrics, the results of 113 pole ligations which we have performed and analyzed postoperatively, and in 66 per cent of them there was a gain in weight, a drop in pulse, and an improvement in the nervous system. When a patient is so ill that you think a fatality is a possibility, and you have an operative procedure as minor as bilateral or single pole ligation which will make this amount of improvement, that is in two-thirds of the cases, then certainly we should not give up pole ligation.

There are one or two other points. One is that low blood cholesterol indicates severe hyperthyroidism. Another point is that we have thought, up until recently, that high blood iodine indicated high blood thyroxin. Thyroxin is 65.2 per cent iodine. It is logical to assume that the high blood iodine seen preoperatively, and which comes to normal postoperatively, is probably the iodine fraction of thyroxin. That unfortunately is not certain.

We now know from recent reports of Mr. H. J. Perkin, who has been doing the blood iodine studies in our Clinic, that out of 331 patients upon whom blood iodine studies were made pre- and postoperatively, 70 per cent had high preoperative blood iodine which came to normal postoperatively; that 30 per cent, on the other hand, had low blood iodine, which after operation rose to above normal, and later, when the metabolism came to normal, returned to normal. This, of course, is disturbing from the point of view of blood iodine possibly representing the iodine fraction of thyroxin.

One of the interesting observations in connection with these atypical findings of blood iodine, has been that without knowledge as to the blood iodine in the 70 per cent of these patients having high blood iodine and coming to normal postoperatively, multiple stage procedures were employed in but 17 per cent of the cases, while in the 30 per cent of patients having low blood iodine preoperatively, which went above normal postoperatively, 46 per cent had multiple stage procedures.

One, therefore, must presume that two features suggesting severity of hyperthyroidism are low blood cholesterol and low preoperative blood iodine. Particularly in this latter group of cases, those with low preoperative blood iodine, we must consider multiple stage operation and when we know that in 66 per cent of the patients having preliminary pole ligations, there is a drop in metabolism, a gain in weight, and a drop in pulse rate, and that in approximately 80 per cent of the patients having preliminary hemithyroidectomy followed by second stage hemithyroidectomy, there is a drop in metabolism, a gain in weight, a drop in pulse rate, then surely multiple stage procedures should be kept in vogue for the patient with severe hyperthyroidism.

Dr. John deJ. Pemberton (Rochester, Minn.) closing.—I think Doctor Lahey is perfectly right in stressing the importance of the liver as a factor in the cause of death in these cases, but I do think that the hyperthyroid reaction probably imposes an additional burden which the liver is not able to carry on during the time.

I feel just a bit guilty in even mentioning this form of treatment without having had greater experience in its use, and I want to make myself unmistakably clear regarding one point, namely, that this form of treatment with cortin is not used as a substitute for any of the methods or measures that have proved valuable but is only used as an additional measure in the hope of preventing or checking the reactions.

PERICARDIAL RESECTION IN CHRONIC CONSTRICTIVE PERICARDITIS

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THE cure of chronic constrictive pericarditis by operation has been one of the most satisfactory chapters in the history of the surgery of the heart. The disease is admittedly a rare one, but the fact that three clinics in this country and several abroad have assembled sizable series of cases can mean only that the diagnosis is being overlooked elsewhere. The surgeon is dependent upon his medical colleagues for the diagnosis, as the few cases of the disease are found under the care of internists. They are variously mislabeled as tuberculous peritonitis, cirrhosis of the liver, valvular heart disease, or ascites of unknown origin. Dr. Paul White has presented the diagnostic problem in detail in his St. Cyres Lecture.[1] With the exception of three more recent cases included in the present report, he has presented the case histories in detail. White's historical résumé is of real interest in showing that the establishment of the condition as a disease entity far antedates Pick, whose name is commonly perpetuated as the eponym. The present communication will deal only with the surgical problem offered by constrictive pericarditis.[2] The details of the case histories as summarized by White will not be repeated and the discussion of diagnosis will be limited to certain aspects that bear directly upon the surgical program. In addition, the list of cases here presented does not conform to White's series to the extent that patients not operated upon by the author have been omitted. The results summarized in Table I are therefore to be considered as the personal series of one surgeon rather than a general hospital report. But as all cases entering the Massachusetts General Hospital since 1931, and found suitable for surgical intervention, have been assigned to the author through the courtesy of the staff, the report does not represent a selected group, except as noted.

SELECTION OF CASES SUITABLE FOR SURGERY. *Rheumatic Heart Disease.*—White states that "if rheumatism can cause Pick's disease it does so in only the rarest cases." This statement is in direct contradiction to the impression gained from a study of the case reports on record in the literature, particularly from European clinics. It is based on the fact that no one of the 16 patients under observation with this complaint at the Massachusetts General Hospital gave a history of rheumatic infection as an etiologic factor. As further evidence, White refers to a group of 1,000 children with rheumatic heart disease followed over a period of ten years at the House of the Good Samaritan in Boston. In not a single instance was there evidence of chronic

constrictive pericarditis, although in many of the patients episodes of acute rheumatic pericarditis had been noted.

This fact is of utmost importance to the surgeon and should lead him to question seriously the propriety of undertaking the operation in patients with rheumatic heart disease. While rheumatic infection not infrequently causes obliteration of the pericardial cavity, the evidence that these adhesions may produce the constrictive pericarditis syndrome is slight indeed.

Active Tuberculous Pericarditis.—Active tuberculosis of the pericardium may produce the entire syndrome of chronic constrictive pericarditis. The point at issue is whether operation can be effective if performed during the active phase of the infection. The reports in the literature of operations performed during this period are uniformly discouraging and confirm our personal experience.

We have, on two occasions, attempted to relieve the serious and progressive tamponade encountered as a terminal event in active tuberculous pericarditis. The nature of the pathology in both instances absolutely precluded relief by surgery. The anterior aspect of the thickened parietal pericardium was excised without difficulty. The fluid, that was in itself relatively unimportant in causing tamponade, was evacuated. The heart was small and lying free within the rigid walled cavity formed by the parietal pericardium. Its surface was covered by a dense, acutely inflamed capsule many millimeters in thickness almost completely throttling the diastolic filling of all chambers of the heart. Even a decortication of the ventricles was out of the question. Both patients died promptly, one on the operating table and the other a few hours subsequently, their lives being shortened a few days as a result of the operative procedure. A third patient has been seen recently upon whom operation was not attempted as a result of these experiences. He died within a few weeks with disseminated miliary tuberculosis. Autopsy findings confirmed our impression that decortication of the heart was impossible.

The diagnostic problem is, therefore, to recognize the active phase of the disease and postpone operative interference with the belief that the best chance for life lies in a self-limitation of the active tuberculous infection. If, and when, the activity subsides and the patient is left with a healed scar constricting the heart, surgery can be successfully undertaken. During the active infection the effects of tamponade can be somewhat controlled by repeated aspirations and diuretics. Rest, light treatment, and other general measures may be employed as in tuberculous peritonitis. The value of pneumapericardium or the possibility of oleopericardium under these conditions have not as yet been established.

The probability that many of the cases of chronic constrictive pericarditis represent healed stages of a pericarditis due primarily to tuberculosis is an important question still to be settled. Examination of the scar tissue removed at operation universally fails to establish a diagnosis of tuberculosis. On the other hand, the frequency with which calcium deposits are found, and the

unusual density of the scar tend to link the pathology with the tubercle bacillus.

The only reliable finding that distinguishes the active phase of tuberculous pericarditis from constrictive pericarditis due to a healed scar is the demonstration of the tubercle bacillus in fluid aspirated from the pericardium. The sudden appearance of the syndrome in a patient with known active tuberculosis elsewhere is presumptive evidence. Fever, increasingly severe tamponade, bloody pericardial fluid, rapid sedimentation rate, and other signs of active infection are suggestive, but are occasionally met with in patients not showing active pericardial tuberculosis. In case of reasonable doubt, it is best to make a limited exploration under local anesthesia. Active infection can usually be recognized by the presence of tubercles on the inner surface of the parietal pericardium. Figure 1 demonstrates the reproduction of the disease in a monkey by injection of pieces of pericardium from one of our fatal cases into the pericardial cavity. The tamponade is manifested by the edema of the eyelids and scrotum, and the terminal dissemination of tuberculosis by the tubercles studding the omentum. The pericardium and heart formed a fused mass of tuberculous tissue.

FIG. 1.—Tuberculous pericarditis in a monkey, terminating in general dissemination of tuberculosis. Peripheral edema and ascites.

OPERATIVE TECHNIC. *Anesthesia.* The magnitude of the operative procedure and the necessity for complete control of the situation, should the heart itself be accidentally torn, make a general anesthetic the method of choice. Simpler operations such as the Brauer cardiolysis or exploration of the pericardium by a limited chest wall resection can of course be accomplished with local anesthesia. The likelihood of opening either, or both, pleural cavities makes it desirable to employ a method of administration allowing differential pressure to be established. Any anesthetic agent that can be combined with adequate oxygen concentration is suitable. In our cases this has been ether administered intratracheally.

Position of Patient.—We have employed a dental chair, believing that a semirecumbent position gives a desirable exposure of the field and in addition tends to diminish the venous return to the heart.

Exposure of the Pericardium.—An ample chest wall window is usually attained by resecting the third, fourth and fifth costal cartilages with about one inch of the corresponding ribs. At times the sixth cartilage and rib end are also resected. No attempt is made to do a subperichondral resection of the cartilages as it is desirable to leave a flexible anterior chest wall over the liberated heart. After ligating the internal mammary vessels the margin of the sternum is exposed, allowing a liberal resection of the left half with the Lebsche sternum knife. The left pleural reflection is now mobilized and separated from the pericardium. At times this is so adherent that opening into the pleural cavity cannot be avoided. In one instance the usual "area of safety" to the left of the sternum was completely absent and the pleural reflection extended to the border of the sternum. The reflection of the right pleura can usually be identified in the upper portion of the field as it reaches the left sternal border. In one case the four major serous cavities of the body, both pleural cavities, abdomen and pericardium were opened during the course of the operation.

Removal of the Scar.—When an area of parietal pericardium has been exposed by dissection and retraction of overlying structures, it is incised preferably in the thinnest area that may overlie the left ventricle. A plane of cleavage is established between the heart muscle and the scar. The anatomic position of this plane of cleavage is not always clearly discernible but usually lies between the visceral pericardium and the layer of organized exudate that has attached itself more firmly to the parietal layer than the visceral. It is essential to the success of the operation to select a plane of cleavage that lies close to the heart muscle itself. Grasping the edge of the scar, and exerting traction during the subsequent dissection, facilitates the exposure in the more inaccessible areas.

If the scar extends laterally over the left ventricle this region should be removed as the first step. Excision may be carried as far as the phrenic nerve, but I have in no instance sacrificed this structure.

The sulcus formed by the descending branch of the left coronary artery is apt to be the site of densely adherent scar, and this region should be approached with caution to insure the preservation of this important vessel. In one instance a thickened calcified band 2 Mm. in diameter marked this sulcus. A maneuver that has been advantageous in several instances has been to establish a new plane of cleavage over the right ventricle so that the interventricular groove may be approached from both sides.

A second very adherent region is the right auriculoventricular groove, in intimate association with the diaphragmatic pericardium. In many cases of the disease the mobilization of the auriculoventricular groove has been a crucial step in relieving the obstruction to right ventricular filling. Keith has shown that the free motion of the auriculoventricular groove is an important

physiologic mechanism in the action of the right heart. Every effort, therefore, should be made to free this area if it can be done with safety, and once past it the inferior cava can usually be readily exposed. Actual decortication of the auricle is a hazardous procedure owing to the thin wall of this chamber of the heart. It has been attempted in only three instances.

Dissection of the diaphragmatic pericardium and scar from the heart should be as complete as possible but in no instance has been carried to the left auricle. Here in particular one is apt to encounter plaques of calcification that extend into the substance of the myocardium. A persistent venous oozing from the heart following their liberation may require hemostasis by fine silk sutures. A thick calcified shell requires the use of rongeurs.

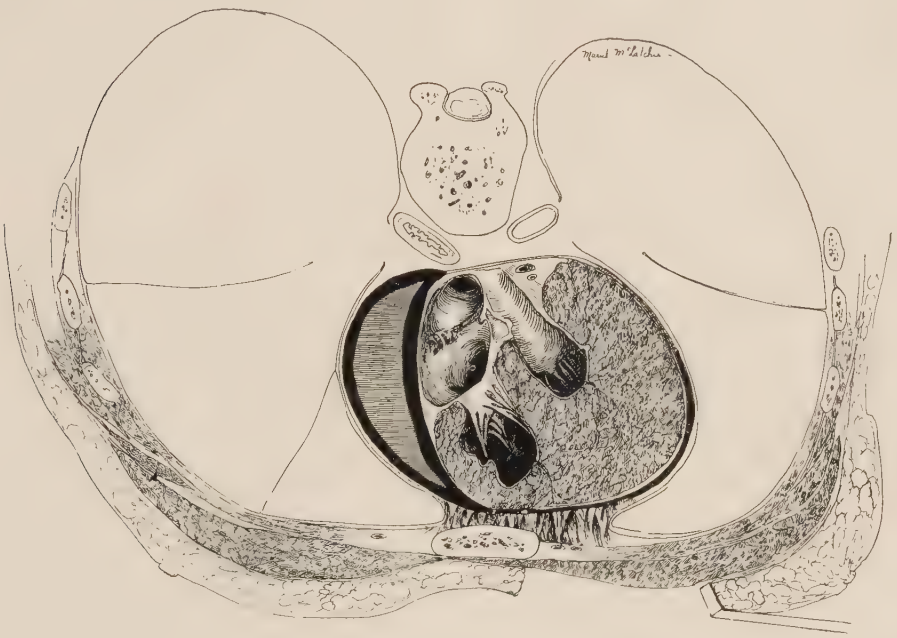

FIG. 2.—Diagram of chronic constrictive pericarditis with encapsulated fluid over right auricle and ventricle.

In general, the outer surface of the pericardium is exposed and bleeding points controlled before freeing the corresponding area from the heart.

Damage to the Heart.—Only in two instances has a chamber of the heart been entered during the operation. In one case a small wound of the right ventricle was readily repaired. In the other case decortication of the right auricle in a good line of cleavage was being carried out by cautious dissection. The tip of a pair of Hartman forceps carrying a cotton pledget plunged into the auricle. A furious hemorrhage resulted that was controlled only by a method suggested by Bigger. As the scar is freed from the heart, a generous flap is always left adjacent to the point of dissection. In such an emergency the scar is replaced on the heart and sutured to close the defect. It was only this maneuver in the case described that averted a catastrophe. In dealing

with a rigid calcified scar even this procedure might be impossible to execute as illustrated by a case reported by Winkelbauer and Schur.[3] Decortication of the auricle should not be attempted in the presence of a rigid calcified envelope.

It is surprising to see how few square millimeters of scar are actually excised in relation to the surface area of the heart that is delivered by the operation. This is due to the increased diastolic filling that is of course the aim of the operation.

Encapsulated Areas of Fluid.—The obliteration of the pericardial cavity may be complete in some areas and residual pockets of fluid be found in others. When an area of encapsulated fluid exists it is important to resect

FIG. 3.—Parietal pericardium and scar dissected from left ventricle. Dense adhesions mark the margin of encapsulated fluid.

the wall of the cavity that is in contact with the surface of the heart. The removal of the parietal pericardium over such an area can be accomplished simply, but will not have the desired effect. A new line of cleavage must be established and a true decortication of the heart carried out. This is illustrated diagrammatically in Figs. 2, 3, 4 and 5, and by the operative sketches as illustrated in Figs. 6 and 7.

Opening the Pleural Cavities.—An opening may deliberately be made into the left pleural cavity if the pleura is densely adherent to the pericardium. The free margin is then sutured to the pericardium at a deeper level. The right pleura may similarly be opened in freeing the right auricle or ventricle. The amount of air that enters may be controlled by positive intratracheal pres-

sure. After the opening is closed the residual pneumathorax may be aspirated with a needle or allowed to remain if the vital capacity is not seriously reduced. The possible advantage of leaving a small pneumathorax will be discussed below.

Closure of the Incision.—After the heart has been adequately freed the muscle and skin flap is replaced to cover the defect and the incision closed. Silk is used throughout to minimize exudative healing. In only one instance has a small drain been introduced to provide an escape for blood. The danger of tamponade from an accumulation of serum does not appear to be great as blood and serum may freely diffuse into the muscle planes of the body wall and are not limited by an inelastic capsule as is the situation in penetrating wounds of the heart and pericardium.

FIG. 4.—Cavity containing fluid is entered and a new line of cleavage developed to separate its inner layer from the surface of the heart.

Postoperative Care.—Transfusion has not been employed and is to be avoided because of the danger of cardiac dilatation from too great a venous return to the heart. If required, citrated blood should be administered very slowly.

An oxygen tent is used routinely after the operation but may be discontinued in a few days.

Drugs, other than diuretics, have little place in the pre- or postoperative program. A syringe containing adrenalin solution should be at hand during the operation for use if cardiac standstill be encountered. The greatest therapeutic safeguard is achieved by maintaining adequate oxygenation both during the operation and subsequently. The heart will tolerate many insults if ade-

quately supplied with oxygen, but withstands poorly any unusual strain or manipulation if attended by anoxemia.

Physiologic Considerations.—Beck has warned against a reduction in cardiac output, that he believes may be of serious degree, produced by the exposure of the heart and great vessels to atmospheric air pressure. This "pneumacardiac tamponade" has been studied extensively in the laboratory by Beck and his colleagues.[4] In conclusion, they recommend a revival of the Sauerbruch negative pressure chamber for operations in which the heart is exposed.

Blalock[5] has been unable to confirm the findings of Beck under slightly modified experimental conditions. Even if the experimental data of Beck be

Fig. 5.—Decortication of right auricle. Parietal pericardium resection unimportant in this area.

accepted as correct, his conclusions appear unwarranted. The experiments with a catheter in the pericardium demonstrating a rise in venous pressure and a reduction in cardiac output following the injection of air are perfectly understandable and were recorded by Cohnheim[6] years ago.

The pressure relations in the experiments with the animal in a negative pressure chamber appear to be confused by the fact that the manometer registering changes in the venous pressure was placed *outside* of the chamber. To prove satisfactorily the point at issue the changes in venous pressure should be recorded on a manometer *within* the negative pressure chamber or cardiac output changes be demonstrated as taking place under these conditions. Such data are not on record.

Subatmospheric pressure applied over the heart and at the same time over

FIG. 6.—Operative sketch showing opening of cavity containing fluid.

FIG. 7.—Dealing with densely adherent area by approach from both sides. Bleeding area controlled with stitches.

the surface of the body (with the exception of the head) is not to be confused with the differential pressure employed in pulmonary surgery. It is difficult to see how differential pressure can be applied to the heart in relation to the rest of the body except by the limited experimental method of a catheter in the pericardium.* If the major portion of the body as well as the heart is exposed to a subatmospheric pressure there is no ground for postulating any force effecting a change in the venous return. Even if the head is kept at atmospheric pressure the theoretical change would be slight as the cerebral circulation is protected by the rigid skull.

These discrepancies are cited to emphasize a diametrically opposite viewpoint regarding the circulation during and immediately after the operation. While Beck focuses his attention upon failure of the peripheral circulation from a *reduced* filling of the heart, I consider the real hazard of the operation to lie in exposing the weakened musculature of the heart after it has been released from the scar to *too great* a venous return. From this point of view the effect of atmospheric pressure on the surface of the heart would be welcomed. I have deliberately attempted to reduce the venous return to the heart during and immediately following the operation. Patients are operated upon in a sitting position under general anesthesia and kept upright during their convalescence. When a small pneumathorax has been accidentally induced at the operation it has deliberately been allowed to remain providing respiratory reserve was adequate. A pneumathorax tends to reduce further the effective venous pressure during the immediate postoperative period.

It must be clearly kept in mind, however, that a reduction in the effective venous pressure may be dangerous if the heart is not relieved of its constricting scar. The high venous pressure in constrictive pericarditis as in other forms of cardiac tamponade appears to aid the heart to maintain an output compatible with life. If this high venous pressure, which in one sense may be considered a compensatory phenomenon, be lowered by loss of blood or by the anesthetic while a high degree of tamponade still exists, the resulting fall in cardiac output may be of serious import. In the cases of active tuberculous pericarditis referred to above, circulatory failure, *i.e.,* inadequate left ventricular output started with the administration of the anesthetic, progressed with the loss of blood during the chest wall dissection, and owing to the fact that the heart could not be liberated, terminated in death. An increment of pneumacardiac tamponade would, it is true, have been a further insult to the circulation under these conditions, but its importance is difficult to evaluate and means to obviate its effect are not at hand.

It may be emphasized again, that the patient will withstand the operation well if the heart is liberated and its muscle is competent. Failing this,

* Sauerbruch's first differential pressure apparatus for animal experiments was designed to do this as it enclosed just the thorax. In his early work with the differential pressure chamber for human subjects the lower extremities of the patient were enclosed in a bag and connected to atmospheric pressure to avoid "venous stasis." This was soon discarded as unimportant. (See Tait: Surg., Gynec., and Obst., **4**, 59, 1907.)

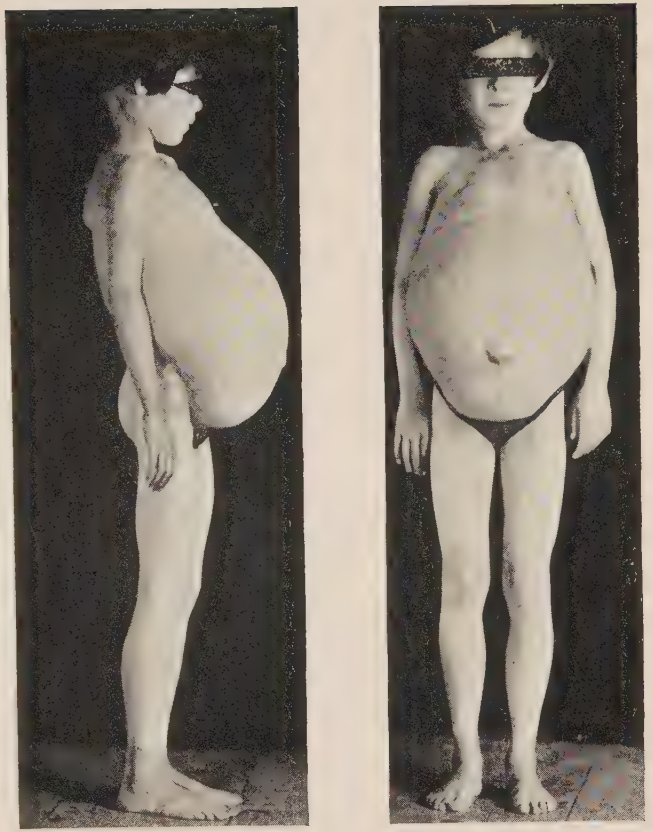

FIG. 8.—Ascites of six and one-half years' duration due to constrictive pericarditis. Unaffected by Talma omentopexy.

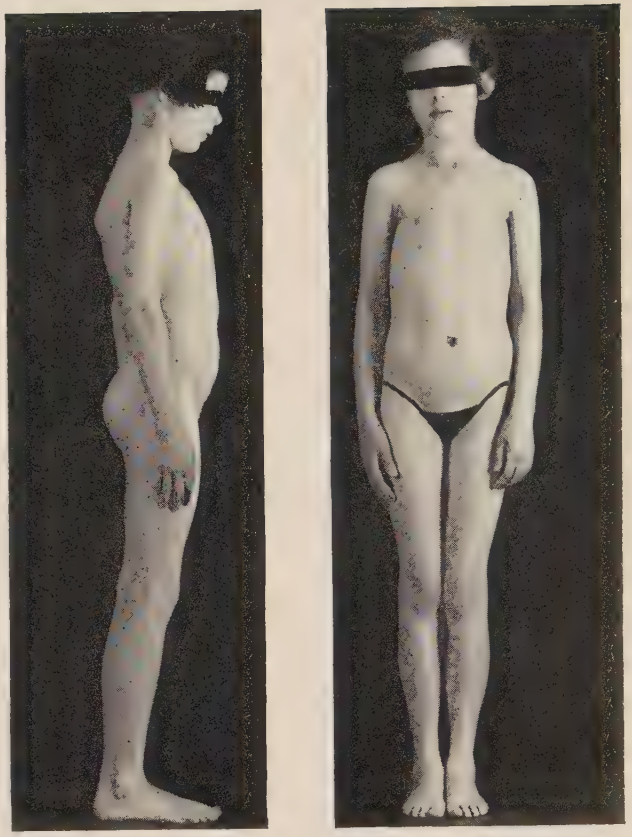

FIG. 9.—Complete cure of patient shown in Figure 8 by pericardial resection.

blood loss or other elements reducing the effective venous pressure may be poorly tolerated.

TABLE I

CHRONIC CONSTRICTIVE PERICARDITIS

Patient	Age	Sex	Date of Operation	Result
1. C. S.	18	F.	7 18 28	Cured
2. J. N.	16	M.	9 27 32	75% improvement. Sedentary work. Persistent ascites
			11 16 33	
3. L. F.	30	F.	4 6 33	Cured
4. C. F.	19	M.	4 22 33	Cured
5. L. C.	12	M.	7 12 33	Cured
6. B. K.	12	F.	11 16 33	Cured
7. D. G.	11	F.	2 27 35	Cured
8. G. P.	43	M.	10 18 35	Improvement. Sedentary work
9. I. B.	47	M.	11 6 35	75% improvement. Light physical work
10. J. H.	52	M.	9 30 33	Died 5th day. Pulmonary edema. Cholelithiasis and advanced portal cirrhosis of liver (not of usual cardiac cirrhous type)
			ACTIVE TUBERCULOUS PERICARDITIS	
11. J. M.	36	M.	4 2 34	Died at operation
12. J. B.	23	M.	1 14 31	Died 12 hrs. postoperatively

RESULTS.—The results are indicated in Table I. By "cured" is indicated normal functional activity for patient's age and sex. In boys this means participation in athletics such as football and track. Complete absence of ascites and edema is also recorded in the cured cases (Figs. 8 and 9). In some instances an abnormal prominence of the neck veins and a moderate enlargement of the liver, the latter due to permanent connective tissue changes, are residual legacies of the disease.

The patients classified as improved show some functional impairment. In one instance this is persistent ascites that has not been relieved by a second and more extended pericardial resection. Functional capacity is greatly improved in this case. The other two cases are still showing improvement of functional capacity and have already been greatly helped by the operation.

Case 10 proved on autopsy to have had an advanced hepatic cirrhosis of the portal type. Portal cirrhosis is not known to be associated with the Pick syndrome and it seems probable that at least part of the preoperative symptomatology may have been associated with this lesion. This patient had a completely calcified pericardium.

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[3] Winkelbauer, A., and Schur, M.: Med. Klin., **31**, 1231 and 1269, 1935.
[4] Beck, Claude S., and Isaac, L.: Jour. Thoracic Surg., **1**, 124, December, 1931.
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[5] Blalock, Alfred: Arch. Surg., **26**, 516, March, 1933.
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Discussion.—Dr. Alfred Blalock (Nashville, Tenn.).—I agree with Doctor Churchill as to the undesirability of operating upon a patient with an acute pericarditis due to tuberculosis. However, occasionally we have found it necessary to do this, because otherwise the compression of the heart would have killed the patient before the chronic stage was reached.

We have at the present time in the Vanderbilt Hospital a patient who was operated upon about a year ago, whose disease was tuberculous in origin, who improved for several months, then became worse again. A second operation was performed a couple of months ago, and the patient is now better but still not cured.

One of the most interesting points connected with the treatment of all patients with constrictive pericarditis has to do with the time at which the operation should be performed, and I cannot answer the question. If one operates during the acute stage, the operation is easier from a technical standpoint, but the patient is more apt to die of the infection. If one operates during the chronic stage the heart muscle is thinned, is atrophic in many areas, and the operative procedure itself is associated with more danger.

Table I shows the findings in ten patients studied at the Vanderbilt Hospital. These patients were observed by Doctors Burwell, Bigger and myself. It brings out some rather striking points in connection with a statement made by Doctor Churchill, namely, that the diagnosis is not difficult, and it is surprising that more cases have not been discovered.

All ten of these patients had distended veins, increased venous pressure, enlarged liver, edema (peripheral), tachycardia, diminished pulsation on fluoroscopic examination and fixation of the heart. Nine of the ten had a paradoxical pulse, low pulse pressure, and faint heart sounds. Eight of the ten had ascites and pleural effusion.

Table I

SYMPTOMS AND SIGNS IN TEN CASES OF CONSTRICTIVE PERICARDITIS

	Per Cent
Distended veins	100
Increased venous pressure (195–390 Mm. H_2O)	100
Enlarged liver	100
Edema (peripheral)	100
Tachycardia	100
Diminished pulsation (fluoroscope)	100
Fixation of heart	100
Paradoxical pulse	90
Low pulse pressure	90
Faint heart sounds	90
Ascites (often early)	80
Pleural effusion	80
Greatly enlarged heart	0
Pulmonary edema (gross)	0
Hypertension	0
Valvular disease	0
Systolic retractions	0
Auricular fibrillation	0
Paroxysmal dyspnea	10

The other findings were not encountered frequently. For example, none of them had an enlarged heart, pulmonary edema, hypertension, valvular disease, systolic retractions or auricular fibrillation. The disease was tuberculous in origin in seven of these cases, and pyogenic in the others. Eight were operated upon. Three are now able to do a full day's work, two are improved, and three are dead. One of the latter three was improved for six months and then died of miliary tuberculosis.

Dr. Edward D. Churchill (Boston, Mass.) closing.—In regard to the recognition of the active phase of tuberculosis of the pericardium, I can add little more to the statement that finding tubercle bacilli in the aspirated fluid is the only certain criterion.

I have purposely specified the active phase of tuberculous pericarditis, because we do not know the nature of the original pericarditis, in many of the so called chronic constrictive cases. It is believed that many of them are healed tuberculosis, but examination of the scar shows no evidence of tuberculosis.

TUMORS OF THE CHEST WALL

KELLOGG SPEED, M.D.
CHICAGO, ILL.

THIS type of new growth is limited to tumors taking origin in the chest wall or the layers of the wall, excluding superficial tumors of the external surface, such as lipomata and superficial cysts of the skin or its appendages as well as all primary tumors of the breast. The proportion of chest wall tumors to tumors of the body as a whole is small. Their frequency may lead to inaccurate diagnosis and prognosis, while their size and situation lend unusual interest. A study of their individual course may necessitate years of observation and their surgical removal may involve both thoracic and abdominal cavities as well as the vital structures contained within them.

Collections of the reports of these tumors have been made by Hedblom,[11, 12] Heuer[13, 14] and others, and now that over 300 instances have been cited in surgical literature, no attempt is made to review the group numerically. The study of the collection of tumors in the Registry of Thoracic Tumors will eventually lead to authoritative statistics easily tabulated for reference. It is my purpose to cite personal experiences with some patients followed for several years.

In 1921, Hedblom[12] collected 213 instances of chest wall tumor; not all primary, however. Of this collection, 167 were tumors of the ribs (78.7 per cent) of which 62.8 per cent were sarcomata and 19 per cent were chondromata. Out of the total number, 46 were tumors of the sternum, of which 54.3 per cent were sarcomata and 13 per cent were chondromata. A later report by him[11] (1933), which covered a collection of 313 case histories, gave 22 personally handled instances to be added to the collection of 78 reported in 1921, and showed that 12 were metastatic tumors which could not be included in the primary group. Their study, however, proved of great value in establishing differential diagnosis, establishment of their identity as secondary tumors being difficult. Of this group, 261 (81 per cent) were tumors of or about the ribs, and 52 (20 per cent) were tumors of the sternum. Of the 70 instances of the rib tumor, 50 were sarcomata and 10 chondromata.

Tumors on the right side of the chest predominate. A history of trauma was obtained in about 20 per cent of the cases and may be an etiologic factor. A favorite tumor site is the anterolateral chest wall. Males predominate (60 per cent), and the ages of patients vary from a few months to 80 years.

Many pathologic types are found among the primary tumors, most of the secondary tumors being carcinomata, metastatic from bronchogenic, adrenal, thyroid or mammary cancer. One classification of primary tumor of the chest wall, advanced by Zinninger,[30] is as follows:

(A) Tumors arising from the deep structures of the thoracic wall partly intrathoracic.

(B) Tumors arising from more superficial structures of the thoracic wall, but apparently fixed to deeper structures.

(C) Tumors arising within the thorax presenting through the thoracic wall.

Subcutaneous fibroma and lipoma and small symptomless chondroma of the ribs, found at physical examination, are not included in this classification. This, I believe, is unfortunate because several instances of malignant tumor, among the reported cases, were traced to very humble origin as small chondromata observed over a period of years, finally becoming malignant. Zinninger reported seven cases from the Peiping Union Medical College Hospital, one case from another Peiping hospital and five cases of superficial tumor and three of intrathoracic tumor presenting through the thoracic wall. These were divided into the following:

Group A.—Eight cases, three radically resected. One died, one recurred four months after resection. The results were not known in three, two presented no change after operation, one was well one year later.

Group B.—Five cases, three operated upon. All apparently cured.

Group C.—Three cases. None operated upon.

Lipomata and hemangiomata of the chest wall may lead to great difficulty in diagnosis and treatment and must be considered in any classification, although they may be quite benign. A large subpectoral lipoma, of 14 years' duration, weighing 3.8 kilos, was removed by Vielle and Eysseric[29] from a man 70 years old. There was no recurrence after removal. Thoracic lipomata have been classified by Heuer as follows:

(1) Hourglass or dumb-bell type.

(2) Anterosuperior mediastinal lipoma presenting at the root of the neck.

(3) Intrathoracic lipoma.

I have recorded an instance of Class 2 which became malignant and inoperable. Case 6 of this report may be of the first type, but no biopsy was performed.

Hemangiomata of the chest wall have been reported by Sorrel,[26] Adams,[1] Lyle[20] and others. Sorrel's patient was a boy, aged ten, whose left chest bulged as if from an intrathoracic tumor extending through the ribs, but both roentgenologic and physical examination failed to give satisfactory information. The mother said that the child had been operated upon soon after birth for a tumor in the chest and there were scars found over the fourth and fifth interspaces with an angiomatous spot near there several centimeters square. The whole chest wall appeared thickened and edematous, without axillary adenopathy. There was some evident increase in the collateral venous circulation seen externally on the thoracic wall. This child received no treatment, although various injections and roentgen therapy were suggested.

Adams' case was a girl, aged five, who had a large tumor on the right chest at birth, when she weighed six pounds and 11 ounces. This had been aspirated and when seen at the later age was an hemangiomatous mass, sessile, lobulated and tense, situated at the right midaxillary line. The mass finally broke

down and ulcerated, after which the patient developed a fever and the growth slowly disappeared, possibly as a result of infection and thrombosis. No operation was performed.

Lyle's case was a woman, aged 77, who presented a swelling in the right, second intercostal space close to the sternum. It enlarged when she bent forward. Operation disclosed a shirt stud hemangioma with expansion between the pectoralis major and intercostal muscles. It was excised en masse. The stem of the collar button mass was composed of a dilated varix, the deeper portion communicating with vessels of the thoracic cavity.

One instance of cystic hygroma of the chest has been recorded by Ingram[15] in a female child, six weeks old, who at birth had a lump the size of a hen's egg in the right chest. This was round and seemed to be free in the subcutaneous tissues. It was removed surgically and found to be a thick walled cyst, with a few small cysts. There was no evidence of malignancy.

The case I reported in 1930 was a malignant desmoid tumor and is included as Case 1 in this report. Cystic lymphangioma of the chest wall has been recorded.

Most thoracic tumors are chondrosarcomata or fibrosarcomata and many undergo myxomatous degeneration. They grow by close infiltration; they metastasize often late and are practically all potentially malignant, even if a microscopic section of a small tumor in an early stage appears benign.

The symptoms are tumor mass, pain, loss of weight, cough, dyspnea, cyanosis, pleurisy and changes incidental to metastases involving the spinal cord or other structures. There may be pleural exudate.

The diagnosis may be evident at sight, especially if the mass has been present for years. The chondromata and fibrosarcomata tend to be fixed to adjacent rib and cartilage and rather steady in growth. Their surface may be nodular and hard. The source of the tumor may be difficult to ascertain, as a flat roentgenogram of the chest may show no changes in rib or sternum, no new bone formation, but a denser shadow than normal for lung and pleura, if the thoracic cage has been invaded. Metastases to the lungs come very late, but a shadow of advancing tumor may be seen beneath the chest wall in lateral or oblique film. Where pleural exudate is present, the roentgenologic findings may be very obscure and misleading as the fluid masks all other shadows. Key[16] said in 1921: "Even in the case of large tumors of the chest wall, the induction of artificial pneumathorax with subsequent x-ray examination and thoracoscopic examination may be of great value for a more accurate diagnosis and for the determination of the extent of the tumor. By such a procedure valuable information may be obtained, which successfully completes the x-ray examination made previous to the induction of pneumathorax."

This advice should be followed in every case where the full extent of the tumor cannot be determined and may be supplemented by the use of the thoracoscope to determine the presence of adhesions between the pleural surfaces, metastases to them or for the purpose of obtaining biopsy material from

within the thoracic wall. Very small tumors between the ribs may be palpated in physical examination and discovered, although there may be no pain or complaint. In the face of uncertainty, an exploratory incision, a thoracotomy and biopsy are indicated, as there is no doubt, if one reviews the histories of the reported cases, that any tumor of the chest wall should be radically excised during its early stage. Pneumaperitoneum is also a great help in differentiating intra-abdominal lesions or extensions, particularly those involving the liver, diaphragm or subdiaphragmatic spaces.

Differential diagnosis must include tuberculosis and syphilis of the ribs, old fracture of the ribs, nonspecific necrosis of any part of the bones of the thorax, dermoid cyst, abscess, aneurysm, multiple myeloma and the great difference between resectable primary and nonresectable secondary tumor. The physical examination must be thorough and should attempt elimination of a primary tumor, and also include roentgenograms, not only of the chest, but also of pelvis, skull and other suspected bones.

Many primary tumors are removable, but demand a well planned, bold attack with suitable preparation of the patient after a searching diagnosis for the extent of the growth. Many of the lateral wall tumors involve the pleura and diaphragm. In the series reported by Lund, six cases involved operative injury to the diaphragm; all recovered. Lockwood,[19] Speed and others have reported such cases. Hedblom collected a series which contained 12 extensions to the diaphragm, showing an incidence of secondary tumor of the diaphragm in 14 per cent of malignant chest wall tumors. Secondary tumors of the diaphragm from any other cause will rarely be of surgical importance as operation would be useless. The pericardium and sternum are often involved. Primary tumors of the sternum, while rare, may be found or palliative operations for secondary tumors of the sternum may be indicated, as recorded by Graham and George W. Crile, Jr.[6] Pulsating tumor of the sternum is generally metastatic. Out of the total of 18 cases collected from the literature by Crile, nine were metastatic from hypernephroma and the same number came from malignant adenoma of the thyroid. Tumors may arise at the costovertebral junction or may be confused with an active syphilitic infection, as in instances recorded by Heuer.

The dangers of opening the thorax are real at any operation for the removal of the chest wall tumor. A graded operation may be indicated to avoid lung or mediastinal collapse. Hemorrhage may get beyond control and no operation should lead to such abnormal mobility of the chest wall that cardiac and respiratory embarrassment follow. Many operations are followed by recurrences of chondro- or fibrosarcomata, partly because of incomplete local excision, so difficult are the technical problems of removal. The chest wall and diaphragm must be approximated and phrenicotomy may be required either before or after operation to permit apposition of these tissues when partially resected. In some cases, a secondary operation to strengthen the chest wall may be required. Harrington has suggested using the scapula to help cover an opening if the defect is large and posterior.

From the Chest Tumor Registry, Andrus reviewed 155 new growths, 117 of which were well diagnosed; 38 were of unproven diagnosis. Of the chest wall tumors, sarcoma predominated. The literature leads me to believe that chondro- or myxochondrosarcomata dominate in number, but only four out of 16 chest wall tumors in the Registry are so classified, ten being fibro- or osteogenic tumors. There were six males and four females and ages varied from 12 to 60 years. Previous trauma was mentioned in but two of the 16 reports. The results of 12 operations listed in the Registry were: one death from shock in five hours, seven recurrences or metastases in from three months to three and one-half years, and four free from recurrences one to five years after operation. My patient, with a malignant desmoid, still lives, 12 years after operation.

In a search of the literature, I can find but one other instance of cranial metastasis similar to that shown in Case 5, who also had a metastasis in the left thigh, both being outside of bone. In February, 1928, Polloson and Novel[25] recorded an instance in a male, aged 25, with a painful tumor just below the right breast. There was a family history of tuberculosis. His tumor was hard, smooth, attached to ribs, extending from the axillary lymph nodes. Roentgenography showed a quadrilaterally shaped tumor centered at the fifth rib. A puncture was negative. The tumor was removed by operation and proven to be sarcoma. On August 20, 1930, the patient reported showing a metastatic mass in the left frontal and two rounded masses in the occipital region looking like sebaceous cysts, but of very rapid growth. They were accompanied by headache, and roentgenologic examination showed loss of cranial substance at the areas mentioned.

Metastases from malignant osteogenic and chondromatous tumors into the skin, where they form either new bone or cartilage, are most unusual. Case 5 becomes of interest on this account alone.

CASE REPORTS

Case 1.—Female, aged 35, married, one child, first seen in April, 1924. In 1912, she had jumped from a street car and in addition to a possible skull fracture had sustained some injury of the right chest, but there were no known fractures of the ribs. In 1917, a tumor mass had appeared on the anterolateral aspect of the right chest wall over the lower ribs and their cartilages. This grew for five years, reaching the size of a child's head, when an attempt was made by a surgeon to remove it through an incision in the right rectus region extending up onto the thoracic wall. It was impossible to obtain from the hospital where this operation was performed any record of the extent of the surgical intervention or the pathologic examination of the tumor.

Examination by me in April, 1924, showed a well nourished woman presenting a tumor the size of a grapefruit on the anterolateral wall of the right chest attached to the lower ribs (Fig. 1). The tumor was rounded, solid, had a consistency of cartilage and extended over the area from the sixth to the tenth rib, inclusive (Fig. 2). The scar of the previous operation, seven inches long, lay median to the main tumor mass extending downward in the right rectus region from the costal margin. Routine laboratory examinations of the patient were normal. The roentgenologic examination showed no apparent pathologic changes in the ribs or other bones. The left side of the chest

showed slightly denser than the right, but there were no evidences of metastases in the lungs. The right diaphragm appeared bulged upward.

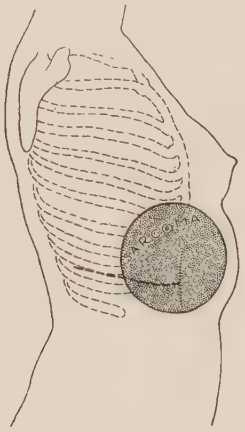

FIG. 1.—Schematic drawing representing the position of the tumor mass on the lateral anterothoracic wall of Case 1. The interrupted line represents the incision at operation. (Surgical Clinics of North America, W. B. Saunders Company.)

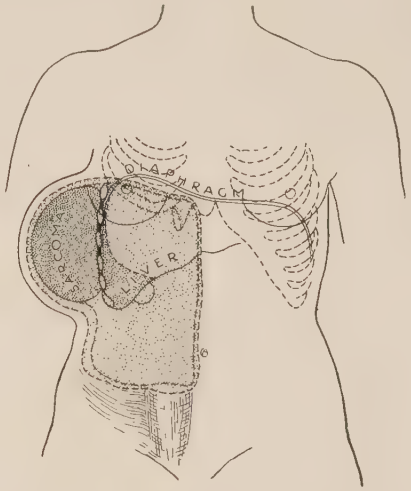

FIG. 2.—Schematic drawing of the position of the tumor mass of Case 1, illustrating its infiltration around the sixth to tenth rib into the liver and diaphragm. The stippled area of the abdominal wall represents the amount of muscular and fascial tissue which had to be removed with the tumor. (Surgical Clinics of North America, W. B. Saunders Company.)

FIG. 3.—Removed tumor of the thoracic wall split open through the central rib enclosed in the mass. Liver substance lies centrally and the thoracic wall in the outer surface of the specimen. At the bottom is some of the infiltrated diaphragm. (Surgical Clinics of North America, W. B. Saunders Company.)

Operation April 28, 1924. Through a transverse incision 12 inches long at the right costal margin extending from the midline laterally and outward, a dissection of the skin away from the tumor mass was performed. To obtain the tumor intact, it was found

necessary to remove all muscular layers of the right abdominal wall down to the iliac crest and backward to the posterior axillary line, including part of the right rectus muscle and the mass of the right lower ribs and their costal cartilages. Both the right pleural and peritoneal cavities were widely opened. No adhesions were found in the right pleural cavity between lung, pleura and diaphragm. The right lung retracted and occupied about half the space of the right side of the chest. To lift the tumor away, it was found necessary to resect the anterior and lateral insertion of the diaphragm for at least ten inches, and in the abdominal cavity the resection was carried through adherent

FIG. 4.—Roentgenologic view taken laterally through the removed specimen of Case 1, showing five ribs, tumor and part of the liver. The main mass has been split to obtain blocks for histologic study. There are no noticeable changes in the osseous structures of the ribs which have been surrounded but not invaded by the new growth of the tumor. The density of the hard fibrous-like tumor is not so great as bone. Desmoid. (Surgical Clinics of North America, W. B. Saunders Company.)

omentum, gastrohepatic ligament and the anterolateral surface of the right lobe of the liver. The liver was resected with the electric cautery, bleeding being controlled by deep mattress sutures of catgut (Figs. 3 and 4). A narrow gauze pack was inserted down to the cut liver surface and the free, retracted edge of the right diaphragm was sutured to the remnants of the transversalis fascia and the sheath of the rectus by pulling the diaphragm well down over the dome and anterior surface of the liver. To close the pleural cavity, the subcutaneous tissue of the chest skin flap was sutured to the diaphragm. The skin was then closed over the abdominal cavity with no peritoneum or muscular tissue remaining beneath. A tubular drain was inserted laterally in addition to the gauze drain extending from the liver surface.

The shrunken gross specimen after hardening was $12 \times 10 \times 8.6$ cm. It included portions of the sixth to the tenth rib, inclusive, the deeper layers of the abdominal wall adjacent to the peritoneum, liver and diaphragm. Histologic examination by Dr. E. R. LeCount gave a diagnosis of desmoid tumor (fibrosarcoma). There were fibroblasts, fibrous tissue, veins with no apparent walls and arteries with no muscular fibers in their walls. There was no direct invasion of adjacent structures by the tumor, but a simple pushing aside or compression with adherence by expansile growth.

The patient recovered without incident or unhappy complications, except for a persistent hernia in the site of the tissue removed. The diaphragm later was studied

roentgenologically and found to regain normal muscle tone and apparent strength, holding its attachment to fascia and the remaining chest wall, not deforming and apparently enervated normally.

The patient has borne four children since this operation. Late in 1935, she underwent an hysterectomy.

Examination March, 1936, 12 years after resection of this desmoid tumor, her age now being 47, shows that her weight is 210 pounds. She appears robust, does all her own housework and runs a small store. General physical examination reveals nothing more than the scars of operation, a recent suprapubic midline scar following hysterectomy and the two long, crossing scars, now white, following resection of the chest and abdominal wall 12 years ago. The ribs are lacking up to the sixth on the right side from the sternum to the midaxillary line. There is a large bulging herniation in the right abdominal and chest area on coughing, but no pain or bowel disturbance and no evidence of tumor recurrence. The liver is not distinctly palpable, although the skin wall over it is thin. Her only complaint is against wearing a corset belt to control the lateral abdominal bulging (Figs. 5 and 6). (This case was listed with the Registry of Thoracic Tumors and reported in Surgical Clinics of North America **10**, 213, April, 1930.)

FIG. 5.—Photograph of abdominothoracic wall and hernia of Case 1, 12 years after removal of tumor.

FIG. 6.—Lateral view of abdominothoracic wall showing extent of scar and hernia.

Case 2.—Female, aged 34, married eight years and has borne three children. In February, 1930, she presented a tumor in the right axillary region of two years' duration with rapid enlargement in the two months before coming to the hospital. The mass had never been painful or tender, there was nothing wrong with the right breast and there was no history of trauma. The general physical and laboratory findings were normal, except for the presence of a firm mass the size of a grapefruit in the right axillary region. This was not reddened nor did it feel hot and it was quite well demarcated, seemingly adherent to the deep fascia and muscles. Roentgenologic examination showed no apparent pathology in the upper half of the right humerus, the chest or pelvis. An exploratory puncture obtained a small amount of thick mucoid fluid tinged with blood and a diagnosis of axillary cyst was made. The tumor was removed February 17, 1930, and the wound healed kindly. The patient left the hospital March 8, 1930.

The specimen consisted of a mass $18 \times 9 \times 6$ cm., attached to an adherent piece of skin 6×4 cm. The portion adjacent to the skin was composed of a colloidal-like substance with evidence of old and new hemorrhage into it along with extensive regressive changes. The cut surface of the main tumor was gray-white and homogeneous, showing on microscopic section fibrosarcoma invading the chest wall muscles.

The patient appeared a second time October 24, 1930, complaining of swelling in the right axillary region for the previous three months with considerable pain for two weeks. She had lost four pounds in weight. Examination at this time showed a mass about the shape and size of a human breast. There was some reddening of the central portion and in the middle was a small area of ulceration, less than one centimeter in diameter, which oozed a little blood (Figs. 7 and 8). This mass was quite hard, fixed to underlying ribs and extended up into the axilla, in which there was no adenopathy.

Roentgenologic examination placed this tumor in the soft parts of the chest wall and no rib or bony involvement was found. In the superior mediastinal area there was an abnormal shadow, which the roentgenologist considered to be a substernal thyroid. It was probably a metastasis of the chest wall tumor.

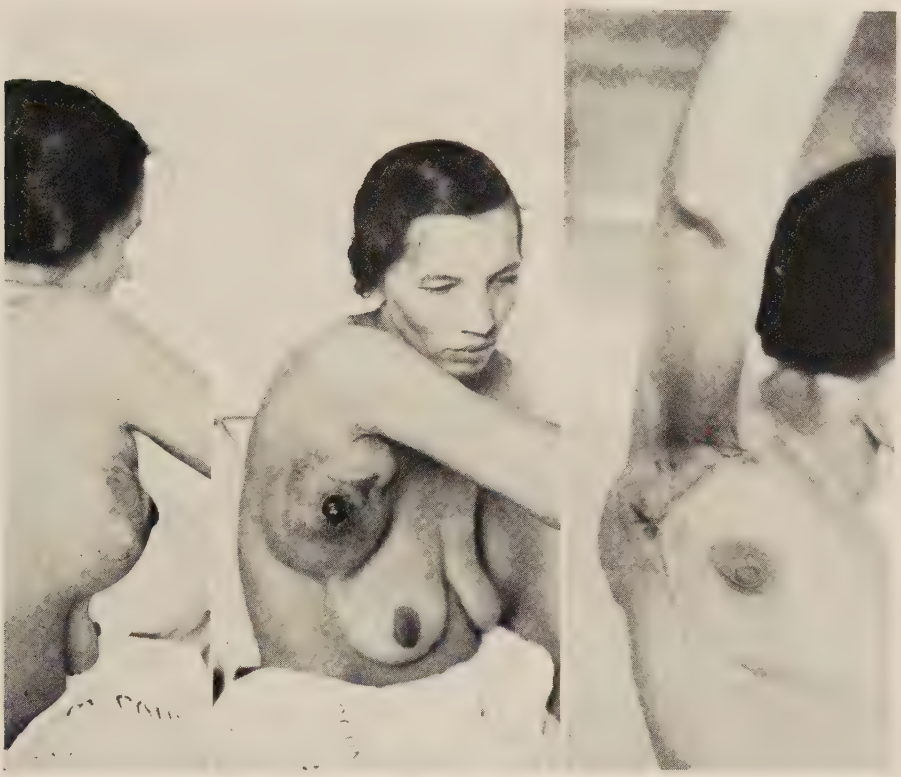

FIG. 7. FIG. 8. FIG. 9.

FIG. 7.—Posterior view, just before operation, of Case 2, showing tumor mass, necrotic center area and pendulous breast below.

FIG. 8.—Lateral view of Case 2, just before operation.

FIG. 9.—Lateral view of Case 2, six months after operation for removal of tumor. The breast has been swung up to cover the defect. Full abduction of the arm is possible.

On November 6, 1930, the tumor mass was removed, together with portions of four underlying ribs, without opening the pleura. To cover the rather large defect in the chest wall, the pendulous right breast was undermined and swung up into the axillary space. This fitted well and permitted wound approximation without undue tension. A second histologic diagnosis confirmed the first, made elsewhere, of fibrosarcoma with marked myxomatous regressive changes.

The wound healed normally (Fig. 9). The patient died one year after operation, from metastases apparently in the mediastinum and lungs.

Case 3.—Male, aged 30, single, first seen in April, 1930. Four months previously, after a sudden jerk of the left arm, he noticed a swelling and some persistent pain over the left upper ribs. The tumor mass, which had developed over the upper left ribs, increased in size, became more painful and he also noticed pain in the right knee. In the four months he had lost five pounds in weight. There was no cough, dyspnea, cyanosis, expectoration or hemoptysis.

Examination showed the chest wall developed; no enlargement of the thyroid. The lungs were normal to percussion and there were no râles. The heart was enlarged. Over the left first rib was a mass the size of an orange, firmly adherent. There were some enlarged left axillary lymph nodes. A diagnosis of malignant tumor of the chest wall, with metastases in the left axilla and right knee, was made. A biopsy on the

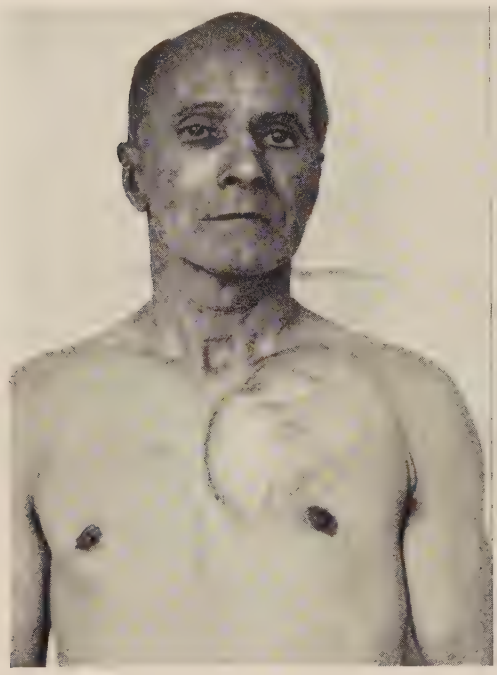

FIG. 10.—Anterior view of tumor of Case 4.

FIG. 11.—Lateral view of tumor of Case 4.

tumor was performed. The patient explained that his first physician had likewise performed a biopsy and told him he had a sarcoma. Roentgenologic examination May 15, 1930, revealed destruction of the anterior portion of the left first rib, but whether this was the site of origin of the tumor could not be definitely stated. On May 31, 1930, a second roentgenologic examination revealed a questionable destructive or lytic process involving the internal condyle of the right femur. No decision was reached as to the character of this osseous change because his knee pain was located over the external condyle of the femur.

Operation for radical excision could not be performed on the chest, because the site of the biopsy had become acutely infected. An histologic diagnosis of anaplastic columnar cell carcinoma was made, but it could not be decided that this was bronchogenic in character. The infection in the tumor continued until June 21, 1930, when the patient left the hospital to go to his home in Porto Rico, no definite diagnosis having been established.

This case illustrates the difficulties of diagnosis between primary and secondary tumor, even after biopsy is performed. Additional examination, including bronchoscopy, or lung collapse, might have determined the source of this tumor.

Case 4.—Male, aged 62, was admitted to the hospital March 31, 1930, complaining of a tumor mass on the left chest wall of three years' duration. When first noticed, this was the size of a pea at the level of the third intercostal space, firm, not tender, gradually increasing until it reached the size of an orange. About six months before he was examined, the mass took on rapid growth and doubled in size until it reached its maximum (Figs. 10 and 11). There was no discomfort or pain.

A general physical examination revealed little abnormal. He was edentulous, but well nourished. There were no lymph nodes in the neck or axilla. Roentgenologic examination showed the main mass of this tumor to be faintly radio opaque; it appeared to be situated mostly on the anterior side of the upper ribs, which were not grossly deformed. A small bulging shadow was also seen in the anterior mediastinum and beneath the clavicle fused with shadow of a sclerotic aortic arch. No metastases were seen in the lungs. The tumor mass was very firm, lobulated, fixed to the chest tissues, encroached on the sternum and extended above the clavicle. A clinical diagnosis of chondrosarcoma was made.

Operation March 13, 1930. A transverse incision over the tumor mass permitted retraction of part of the pectoralis major muscle, the fibers of which were split and the tumor exposed down to its attachment to the second, third and fourth ribs. In attempting to free the tumor, its major portion broke away. The smaller, remaining portion was removed with portions of the attached ribs and costochondral junction clear to the sternum. This excision broke into the pleural cavity and the collapsed left lung was seen. Bleeding points were controlled and the skin was closed tightly over the defect. There was a slight rise in temperature, lasting but two days, followed by a small amount of fluid in the left thoracic cavity and a smooth postoperative recovery, with a discharge of the patient from the hospital March 28, 1930.

The specimen consisted of two tumor masses with attached muscles, one $15 \times 9 \times 7$ cm. and the other $8 \times 7 \times 3$ cm. The larger mass was moderately firm but felt cystic. On gross section it was found to be composed of cyst-like spaces with cartilaginous walls. The cystic areas were filled with glairy mucoid substance and amorphous yellow fibrous-like material. The smaller tumor, with attached ribs, was firmer and more homogeneous. A frozen section revealed a chondromatous structure, but no malignancy could be identified. The paraffin section showed chondrosarcoma with myxomatous degeneration.

He was reported to be alive six months later, but suffering from pulmonary complications and local recurrence. There was free fluid in the chest cavity. No further trace of him has been obtained. The histologic diagnosis was chondrosarcoma.

Case 5.—Male, colored, aged 40, single. Admitted to hospital in August, 1930, complaining of a tumor mass on the right side of his chest (Figs. 12 and 13), which was of five years' duration. The condition apparently originated as a nodule at about the level of the right third rib not far from the sternum. Within a year the mass had reached the size of an orange and was hard and fixed to the chest wall. In 1927, biopsy was performed and he was told the tumor was a fibrosarcoma, its removal advised and operation performed elsewhere. Within one year it had recurred, grown to its size before removal and continued to increase in spite of all treatment.

When first examined in 1930, there was pain in the chest, no cough, no dyspnea, he was doing manual labor and was chiefly distressed on account of the size of the mass. He was able, at that time, to rest his chin on the bulging tumor. The mass was the size of a small melon, felt tensely cystic in some areas and was definitely nodular but did not pulsate. The skin had not broken down over it. His heart was not displaced. His liver was enlarged two fingers' breadth below the costal margin. The Wassermann reaction was negative; blood and urine were normal. Roentgenologic examination

FIG. 12. Anterior view of Case 5, just prior to operation in 1930. The scar of a previous operation is seen, but is not broken down.

FIG. 13.—Lateral view of tumor of Case 5 on same date as Figure 12.

FIG. 14.—Case 5, nine months after operation. Wound healed, no evidence of recurrence yet visible.

showed an opacity in the upper two-thirds of the right chest, extending one inch above the clavicle and covering the axillary region. A lateral view showed a cup-like depression within the chest cavity, centering behind the right third and fourth ribs anteriorly, with a definite margin, no collapse of the right lung and no metastatic masses in the lung areas.

Operation September 11, 1930. A block dissection of the tumor, including the anterior portions of the second, third and fourth ribs with their cartilages, the right half of the sternum adjoining and the edge of the pericardium, was effected. As the tumor was lifted away with the chest wall, pale jelly-like material was expressed from the mass. Behind the tumor lay an intrathoracic cavity as large as a fist, lined with thickened pleura, as indicated in the roentgenogram, and the partly cut away covering of the pericardium. Each heart impulse could plainly be seen. This thickened pleura was not opened as it was too extensively infiltrated and the cavity seemed to extend down to the diaphragm, containing much mucoid-like material which was gently scooped out. There was one enlarged lymph node in the right axilla. A cigarette drain was inserted in the chest pocket and the skin was closed over the defect. Some fever persisted for ten days after operation, but no untoward complications followed and the wound healed.

The tumor mass, after removal, was globular and measured 20 × 16 × 10 cm., partly covered by a skin flap measuring 14.5 cm. wide × 20 cm. long. At one angle of this skin the breast remained unchanged. The tumor was firm throughout, assuming irregularly the consistency of cartilage. The cut surface was variegated with softened, deep-purplish and red areas up to four centimeters in diameter, the entire surface being glossy and translucent. No new bone was found, but cartilaginous areas were evident. The ribs and sternum showed erosion, but little bony reaction. An histologic diagnosis of chondromyxosarcoma with moderate irregularity of the cells and regressive changes, mostly in the form of mucoid degeneration, was made.

The tumor remained quiescent and giving no symptoms, he returned to his work (Fig. 14). On May 19, 1933, a few nodules were formed around the healed scar with some bulging tumor masses, one area the size of a lemon and three or four smaller ones. A sinus had developed in the scar below the level of the second rib, pin point in size, out of which fluid dropped when he leaned forward. This, he thought, was water from his bath, air bubbles being frequently mixed with the discharge. These bubbles were often seen emerging at a rate synchronous with the heart beat. He had gained considerable weight since the operation and was holding it. He felt very well.

Roentgen therapy was then started. In spite of this, the sinus enlarged, its edges became sloughing, gangrenous and ultimately foul smelling. Roentgenologic examination of the chest failed to reveal any metastases in the lungs and no enlargement of the intrathoracic pocket at the site of extirpation. The pulsating pericardium could be seen at the bottom of the opening which had to be dressed several times a day on account of the purulent discharge which could be made to spill over if he bent well forward.

By December, 1935, the condition had progressed until three fingers could be thrust into the chest orifice. The loss of weight and strength became so marked that he gave up work; he also complained of some pain in the left thigh. On examination, there was found to be an enlargement of the soft tissues of the left thigh, rather hard, not bony, attached to the femur beneath. There was also found a nodule appearing on the scalp of the vertex, not painful, fungating in character and bleeding if irritated (Figs. 15 and 16). Roentgenologic examination still failed to show further tumor advance or metastases in the chest. In the left thigh, the metastasis seemed not to be central in the femur, but to be outside of the bone pressing upon it. This was verified by roentgenologic examination which showed no osseous change.

Fearing a pathologic fracture of this bone and realizing the end was near, he was sent to a charity hospital. A biopsy was performed on the tumor mass on his scalp and various parts of the skeleton were rayed. These films showed no evidence of the spread of the tumor or metastases in the lungs and no metastases in any bones. The growths in

the thigh and scalp were extra-osseous. The histologic diagnosis of the metastatic tumors was chondromyxosarcoma. Death occurred March 14, 1936, and no autopsy could be secured.

Case 6.—Female, aged 32, married, seen in July, 1933. Her complaint was a small tumor on the wall of the right side, just above the breast near the midaxillary line. There had been some thoracic pain, but no cough, dyspnea or hemoptysis. The mass was quite hard, was not connected with any palpable or visible mass in the breast, which was normal. The laboratory and general findings were normal.

Roentgenologic examination demonstrated a collar button type of tumor shadow extending between the ribs, the external portion giving a deep shadow beneath the soft tissues without any evident damage of the ribs and the intrathoracic portion, well de-

FIG. 15.—Metastasis in scalp, a few weeks before death, of Case 5. Six years after removal of myxochondrosarcoma of chest wall.

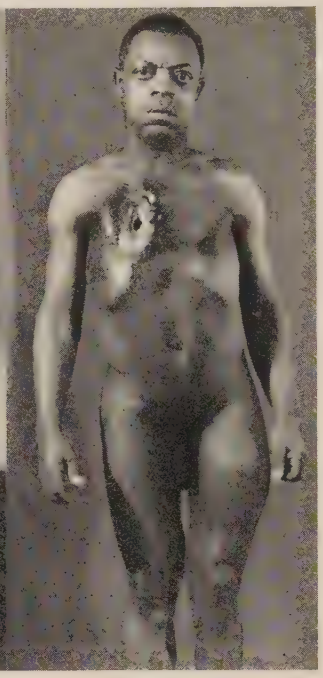

FIG. 16.—Photograph of Case 5, a few weeks before death, six years after the removal of tumor. The sinus leading into the right thoracic cavity is seen surrounded by necrotic scar tissue and neighboring masses of local recurrence of the chondrosarcoma. The small metastasis in the scalp and the larger metastasis in the left thigh, both extra-osseous, are present.

marcated, extending upward and downward into the right thoracic cavity in a semi-pedunculated manner. The inner portion had the appearance of a cyst-like mass with indistinct trabeculae, but was definitely connected with the extrathoracic bulge.

A biopsy and operation were refused and although a definite diagnosis could not be reached, it was felt that the condition was not at that time malignant. The patient did not return after the examination.

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SOME ADVANCES IN THE TECHNIC OF THORACOPLASTY

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IMPROVEMENTS in the technic of thoracoplasty during recent years have more than doubled the percentage of patients whose tuberculous pulmonary cavities have become completely closed, and have decreased by more than half the percentage of deaths that occurred with the Wilms-Sauerbruch paravertebral and the Brauer subscapular types of thoracoplasty.

Although a large number of factors, with respect to operative technic and pre- and postoperative management, have contributed to this almost phenomenal improvement in results, only the most important will be considered in this article. The improvement has not been due to the exclusion of poor risk cases since the division of the modern operation into multiple small stages, as advocated by Alton Ochsner and Hedblom more than ten years ago, has resulted in the employment of thoracoplasty for many patients who could not have withstood a one stage or a two stage operation. The present extensive use of a thoracoplasty that is restricted to the upper part of the chest without a complementary permanent phrenic paralysis has greatly extended the indications for thoracoplasty and has made bilateral collapse therapy possible for certain forms of bilateral cavernous tuberculosis.

The restriction of every stage of a thoracoplasty operation to the removal of two, or rarely if ever more than three, ribs is undoubtedly the most important factor in the reduction of the operative mortality rate. Several years ago when four or five ribs were removed at a stage of the operation, grave cardiorespiratory decompensation, sometimes resulting in death, was a rather frequent postoperative complication. At present this complication, or death from other causes directly or indirectly due to operation, is exceptional. The resection of only two or three ribs at one stage permits the safe removal of great lengths of these ribs, which factor is chiefly responsible for the present high percentage of complete cavity closure. It is, however, obvious that as great sudden pulmonary collapse and dangerous paradoxical motion of the thoracic wall and lung may be produced by the total removal of only two or three ribs as by the partial removal of four, five or six ribs. The surgeon must, therefore, stage the costal resections in the horizontal as well as in the vertical direction, leaving as much of the anterior ends of the ribs as the relative mobility or rigidity of the posterolaterally decostalized portion of the thoracic wall indicates is necessary, in order to preserve a safe degree of stability of the thoracic wall and to prevent too great and sudden a pulmonary collapse. If, after two or perhaps three posterior stages have been performed, roentgenograms show that a pulmonary cavity is being prevented from complete closure by the remaining anterior costal stumps and cartilages,

these may be dealt with by one of the efficient modern types of anterior thoracoplasty operation, which then constitutes a stage of the whole thoracoplasty.

The division of the operation into three, four, or exceptionally five, stages, separated by intervals of approximately three weeks, would result in an incomplete collapse of those parts of the thoracic wall that were first decostalized, if provision were not made to prevent regeneration of ribs from the periosteum. Regeneration can be prevented efficiently if the dried periosteum is thoroughly scrubbed with 10 per cent formalin solution. Since permanent mobility of the decostalized portion of the thoracic wall is not desirable, only those parts of the periosteum that are protected from undue mobility by the rigid scapula and the heavy muscle posterior to, and just anterior to it, should be formalinized unless, as in cases of empyema and some cases of fibroid phthisis, the anterolateral and infrascapular parts of the decostalized thoracic wall are found to be rigid at the time of operation.

The routine removal of the entire lengths of the vertebral transverse processes and the underlying necks of the ribs at, and somewhat above and below, the level of the pulmonary cavity or cavities, greatly increases pulmonary collapse in the costovertebral gutter and has an important influence in averting secondary thoracoplasty. I have an impression that the routine removal of the entire lengths of the transverse processes has, in my clinic, increased the closure of cavities from 10 to 15 per cent.

The old practice of resecting the lower ribs at the first stage of the operation so as to prevent the aspiration of secretions into the lower lung has been largely abandoned since experience has shown that the resection of the upper ribs first is, from a number of standpoints, safer. The removal of the upper ribs first has the additional great advantage of permitting the surgeon to conclude the thoracoplasty as soon as roentgenograms, sputum examinations and other signs show that the cavities have completely closed and that all the important lesions are controlled. In the great majority of cases this condition exists after only six, seven or eight ribs have been removed; the lower ribs are, therefore, preserved for useful respiratory function, which might be invaluable if collapse therapy should at any future time be necessary for the contralateral lung. If a temporary paralysis of the phrenic nerve had been produced before the thoracoplasty had been performed, the later return of diaphragmatic function would obviously add to the respiratory functional reserve.

Emile Holman has recently made the valuable proposal that when the lesions are so limited to the apical portion of the lung that no more than five or six ribs need to be resected, the scapula may be made to fall into the defect in the costal cage so as to produce maximal pulmonary collapse if the inferior portion of the scapula is resected.

Many other factors are responsible for the greatly improved results from thoracoplasty in recent years. Important among them are the use of a 10 or 15 degree Trendelenburg position during the operation and for from 12 to 24

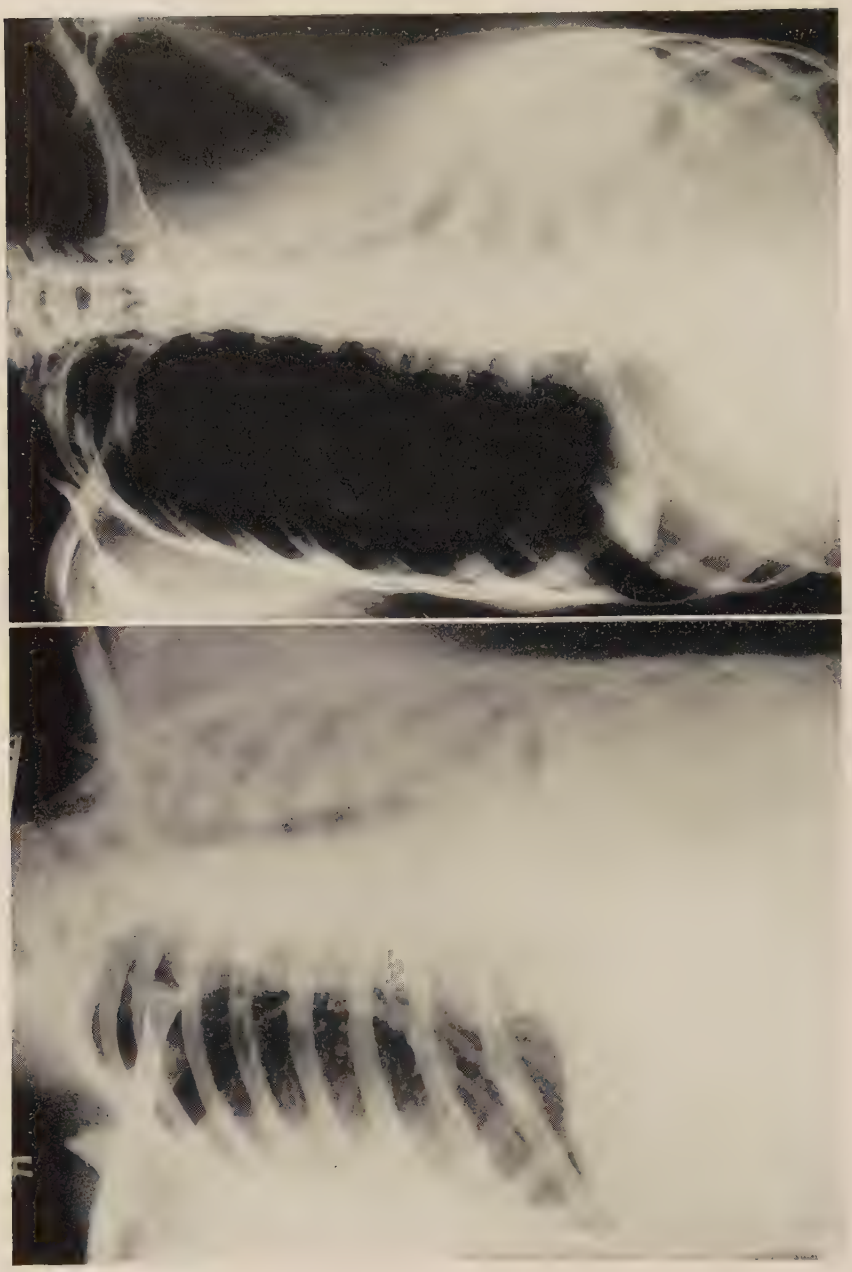

FIG. 1.—Comparison of the pulmonary collapse produced by the old paravertebral and the modern types of thoracoplasty.

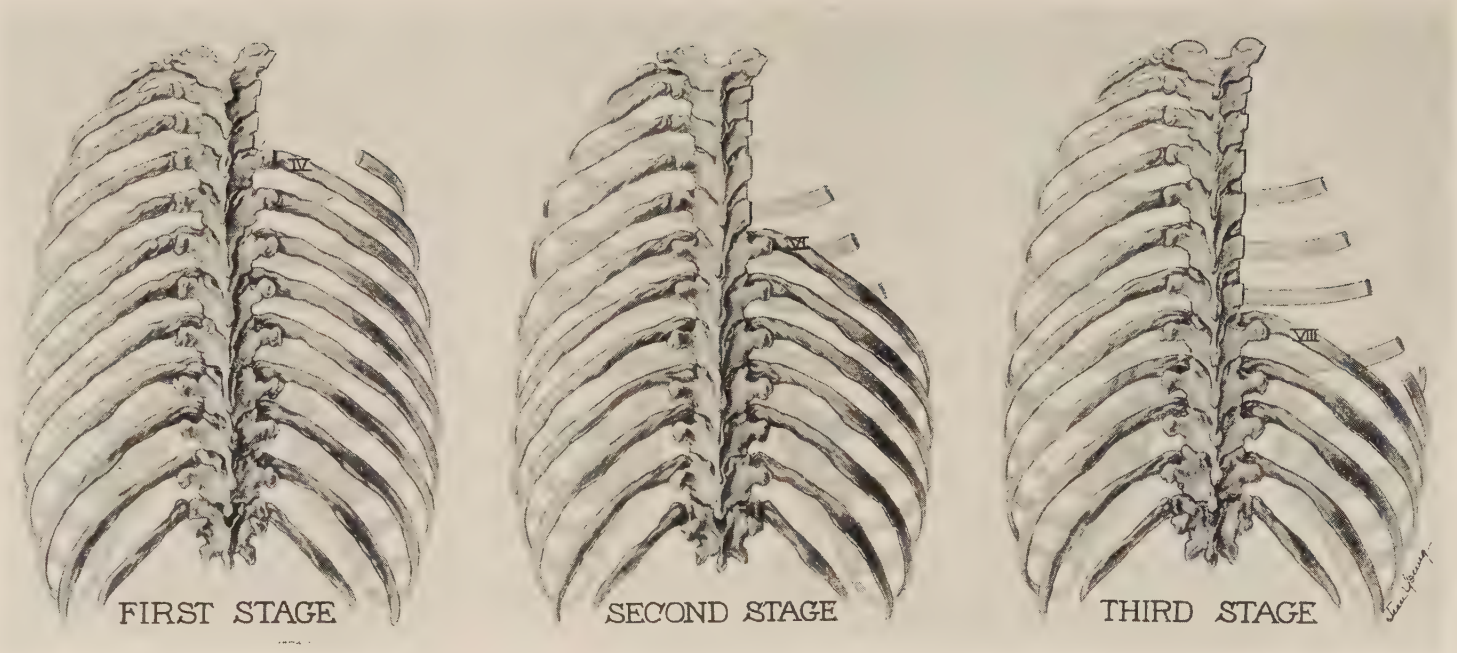

FIG. 2.—Approximate extent of costal resection in modern type of posterolateral thoracoplasty of seven ribs. At the first stage, all the first rib and cartilage, the posterolateral portion of the second rib, and a part of the posterior portion of the third rib have been resected. At the second stage, the remaining posterolateral portion of the third rib and the posterolateral portions of the fourth and fifth ribs have been removed. At the third stage, the posterior, and parts of the lateral portions of the sixth and seventh ribs have been removed. The transverse processes and the underlying necks of the ribs, except the first, have been entirely resected.

hours thereafter, so as to favor the gravitation of pulmonary secretions toward the mouth rather than toward the undiseased dependent lung. Immediately before and after operation, while the patient is in the Trendelenburg position, he should cough repeatedly and expectorate as much secretion as possible. Furthermore, he should cough at least every hour while awake for a day or two after operation so as to prevent the stasis of secretions and the development of pneumonia or new areas of tuberculous infiltration. The routine intravenous administration of 3,000 cc. of 5 per cent glucose solution during approximately the first eight postoperative hours keeps the patient in water balance during this important period, combats a tendency toward low blood pressure, and makes unnecessary the giving of fluids by mouth.

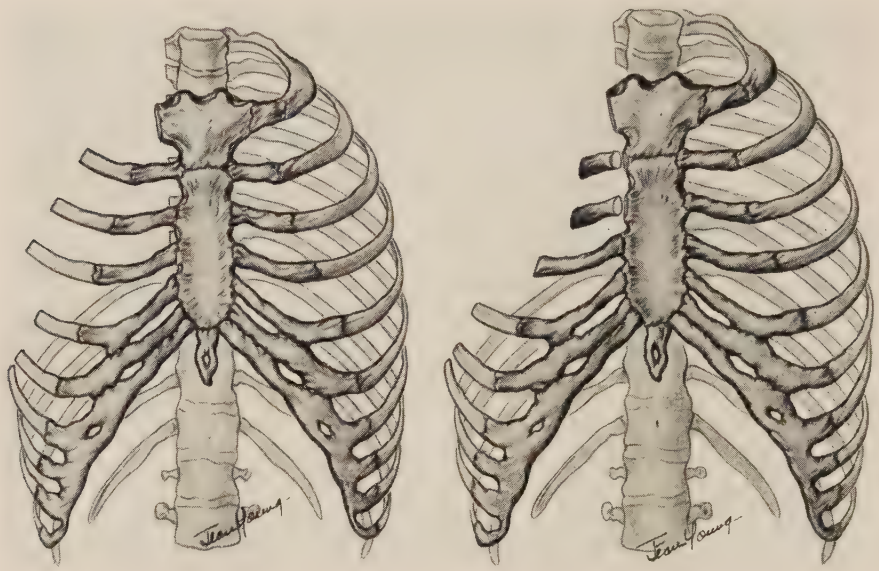

FIG. 3.—Haight type of anterior thoracoplasty. The left-hand drawing shows approximately the lengths of the anterior costal stumps remaining after completion of the posterolateral thoracoplasty illustrated in Figure 2. The right-hand drawing indicates the additional collapse produced by the removal of the second, third and fourth costal stumps and the parasternal division of the second and third cartilages. (From Haight, Cameron: Journal of Thoracic Surgery, 5, 453, 1936.)

In case an ineffective pneumathorax is present when a thoracoplasty is decided upon, the air should be allowed to become completely absorbed before the thoracoplasty is performed so that the danger of an operative opening of the thin, nonadherent parietal pleura, or of a sudden increase of pressure upon the lung and mediastinal organs by the falling in of the decostalized portion of the thoracic wall upon the closed pneumathorax, may be avoided. The gradual expansion of a partially collapsed, active tuberculous lung only occasionally proves harmful and need not be feared unless fever or other signs of increasing activity of the lesions occur, even though cough and expectoration increase and a partially collapsed cavity enlarges. If, however, a recurrence of fever, the roentgenologic finding of an increase in the parenchymal infiltration, or pain, dyspnea and persistent irritative cough from

unduly high negative intrathoracic pressure should occur, no further attempt should be made to cause the lung to expand completely. In such a case pneumathorax refills should again be given until the new tuberculous activity or the signs of unduly high negative pressure have subsided and then the thoracoplasty performed in the presence of the pneumathorax, air being aspirated before, during or immediately after operation so as to prevent unduly high intrapleural pressure. Many other technical factors bearing upon the safety and effectiveness of thoracoplasty might be considered but are beyond the scope of this article.

Haight and Alexander, in an article to be published, report that 119 of their patients have had the modern type of thoracoplasty during the two and one-half year period ending January 1, 1935. In April, 1934, all cavities of these patients were completely closed as indicated by roentgenograms made by the Potter-Bucky technic, and the sputum was continuously negative to concentrated specimen examination in 83.1 per cent, or in 93.4 per cent of the 106 living patients. Only 10.9 per cent of the 119 patients had died by the late spring of 1935 either from operation or tuberculosis; only 5.4 per cent of the 37 patients operated upon in the year 1934 had died by August, 1935. One hundred and two (96.2 per cent) of the 106 living patients have closed cavities in the lung of the side operated upon but three of them have positive sputum from open lesions in the contralateral lung.

In striking contrast to the figures just given are those I collected in 1925 from many clinics in different parts of the world which were using the old type of thoracoplasty: Thirty-six and eight-tenths per cent of 1,159 patients had closed cavities and negative sputa, and 38.7 per cent died a variable length of time after operation. The following figures collected by Hedblom and Van Hazel in 1934 for the 3,762 old and transitional types of thoracoplasty operations performed between 1925 and 1934 are approximately the same: 35.3 per cent practically symptom free and 33.6 per cent dead.

Although the figures cited in each of the preceding two paragraphs are not exactly comparable, since those in the second paragraph represent a longer average time after operation, the difference in the figures approximately represents the difference in effectiveness and safety of the old and new types of operation.

SUMMARY.—(1) Improvements in the technic of thoracoplasty during the last ten years have virtually halved the operative mortality rate and doubled the percentage of patients whose tuberculous cavities become completely closed.

(2) Among the more important technical improvements which have exerted an important influence in extending the indications for thoracoplasty as well as for various types of bilateral collapse therapy are the removal of no more than two or three ribs at any operative stage; the removal of greater lengths of the ribs; if maximal collapse is needed, the anterior ends of the ribs are removed at a separate stage so as to lessen the suddenness of pulmonary collapse and reduce dangerous paradoxical movement of the thoracic wall; provision for progressive pulmonary collapse by the prevention of re-

generation of ribs posterolaterally through formalinization of the periosteum; resection of the entire lengths of the vertebral transverse processes and the underlying necks of the ribs at, above, and below the level of the pulmonary cavity so as to increase pulmonary collapse in the costovertebral gutter; the removal of the upper ribs first and the preservation of the lower ribs for useful respiratory function when there are no lesions in the lower lung requiring collapse. A large number of other factors in the modern thoracoplasty operation have contributed to the reduction of the operative risk and in bringing about complete healing of the tuberculous lesions.

(3) Statistics are cited to show the striking improvement that has occurred in the results of thoracoplasty during the last ten years.

THE LATE RESULTS OF THORACOPLASTY IN THE TREATMENT OF PULMONARY TUBERCULOSIS

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The operation of extrapleural thoracoplasty is well established as the method of treating certain types of pulmonary tuberculosis. However, the technic of the operation has changed considerably in recent years. Formerly, relatively short segments of the first 10 or 11 ribs were removed in all cases, usually in two stages, beginning with the lower ribs. At present, the tendency is toward a partial thoracoplasty involving the removal of very long segments of a few ribs over the diseased portion of the lung, thus preserving the function of the non-diseased areas. The partial thoracoplasty is begun at the upper limits of the thorax and continued downward until the desired amount of compression has been obtained. Sufficient time has not elapsed since the advent of the partial operation to compare the permanency of the results accomplished by it with those of the earlier complete operation. It would seem advisable, therefore, to record the late results in as large a number of cases as possible which have been operated upon according to the earlier technic, in order that a satisfactory comparison can subsequently be made with those resulting from the present procedure. A further reason for making such a report is to attempt to answer the questions: "What are the late results which can be obtained by complete thoracoplasty?" and "How lasting are such results?"

A partial thoracoplasty is commonly defined as consisting of the removal of segments of seven ribs or less. According to this standard, all of the cases in this report have been subjected to a complete thoracoplasty. They have all been operated upon by a technic the details of which have been described elsewhere,[1] and the principles of which are meticulous hemostasis, gentle handling of tissues, local anesthesia, the use of silk for ligatures and sutures and closure of the wound without drainage. In the earlier cases in this series, the operation was performed from below upwards, but beginning in 1932 the upper portion of the thorax was attacked first. In the later cases in the series much longer segments of ribs were removed, over the most diseased portions of the lung, than had been the case in the earlier ones, as it was becoming apparent that many of the cavities had not been closed by the resection of short portions of the ribs. By far the majority of the operations were performed in two stages, with a two week interval between them. At each stage, segments of at least four and frequently of five or six ribs were removed. At present never more than three ribs are removed at a stage, but practically the entire length of each of the first three ribs is resected.

It has been the experience of many surgeons that the resection of short rib segments is ineffectual in closing cavities and is directly responsible for many of the deaths and unsatisfactory results in their earlier cases. Even if a complete thoracoplasty is proposed, it is essential to remove long enough segments of ribs to completely close the cavity. In the 103 cases embodied in this report from two to eleven years have elapsed since the operations. Most of the patients had been ill with pulmonary tuberculosis for from three to five years; the shortest duration of the disease prior to operation was one year, and the longest 12 years. All of the patients had had unsuccessful attempts at sanatorial care and at artificial pneumathorax, and many had had a phrenicectomy performed.

Several different methods of classifying cases in order to indicate the results of thoracoplasty have been used. Probably the simplest is that of Bull[2] of Oslo, who in 1930 reported on 401 cases, some of which were collected. His classification was Group I.—Alive, able to work, practically symptom free, negative sputum; Group II.—Alive, able to do some work but not symptom free, positive sputum; Group III.—Unable to do any work, positive sputum; Group IV.—Results not evident; Group V.—Unable to be traced; and Group VI.—Dead at various time intervals. This classification was used by Hedblom and Van Hazel[3] in their comprehensive report published in 1934, and is the one I propose to use. However, there are a few cases which cannot be fitted into the above classification and I have placed them in a Group I-A.—Alive, not entirely symptom free, able to do very little work but with negative sputum.

The results of thoracoplasty will vary with several factors. The more important ones of these are: (1) the care with which patients are selected for operation; (2) the type of operation performed, and (3) the type of convalescence which follows operation. The more strictly the indications for operation are adhered to, the better are the results. Thus Archibald[4] found that among "good chronics" his operative mortality was 4.3 per cent; in the group of doubtful risks it was 4.2 per cent, whereas among the unfavorable cases it was 26 per cent. Brunner,[5] reporting on 117 cases performed in Sauerbruch's Clinic, obtained 40 per cent cures in a group of cases with favorable indications, 14 per cent cures in cases with less favorable indications and no cures in the desperately ill cases. Table I indicates our experience in this regard.

TABLE I

TYPES OF PATIENTS OPERATED UPON

"Good risks."—62 cases, 59.6% are apparently well—22.6% have died.

"Doubtful risks."—27 cases, 40.7% are apparently well—29.5% have died.

"Unfavorable risks."—14 cases, 30% are apparently well—35.2% have died.

However, it must be remembered that if only the most favorable cases are selected for operation, the benefits of the operation will be denied to many patients, a considerable number of whom can be cured. It is advisable,

on the other hand, not to accept hopeless cases for operation on the plea that it gives them their only chance, for that type of patient will practically always die as a result of the operation. Several of the cases reported in this paper should never have been operated upon, and in the light of our experience would not now be thought suitable for operation. Careful observation by a competent internist, the use of blood transfusions and graded exercise may do a good deal towards converting a seemingly hopeless risk into a moderately good one.

Mention has been made above as to the type of operation which is considered most effective at the present time. Many of the early operations in this series were quite inadequate. As evidence of this are the 13 cases that had to have "supplementary" operations for cavities not closed by their original operation. The time to close the cavity completely is at the first series of operations. Supplementary operations have proved rather unsatisfactory in our experience. It is most important to bear in mind that the operation of thoracoplasty does not cure the patient at once, but that it makes conditions such that nature can more readily heal the cavities and other lesions in the lung. Consequently a sufficiently long and properly managed convalescence is essential following operation. All of the patients included in this report were kept at absolute bed rest for at least six months after operation. If the sputum was still positive at the end of that time, the bed rest was continued up to one year before additional collapse therapy was advised.

In considering the results obtained by thoracoplasty, it should be borne in mind that the patients upon whom the operation is performed are chronically ill and have been so for years, that they are frequently very poor risks and that without the benefits of operation the outlook for all of them is practically a hopeless one.

Table II shows the late results in the patients that fall into Group I, *viz.*, those that are living, are able to work, are symptom free and have a negative sputum.

TABLE II

GROUP I:—ALIVE, ABLE TO WORK, PRACTICALLY SYMPTOM FREE, NEGATIVE SPUTUM

No. of Cases	Years after Operation												
103	2½	3	3½	4	5	6	7	8	9	10	11	Total	Per Cent
	2	6	3	3	7	6	5	10	7	8	1	58	56.3

In Hedblom's report[3] in 1934 of 200 cases of his own, 41 per cent fell into Group I. He also collected 1,235 from the reports of eight authors and of these found that 35.4 per cent were in this group. At least two years had elapsed since operation in each instance. Graham[6] reports that 29.3 per cent of his 75 cases, in which the operation had been performed for at least two years, were able to resume normal activities and had negative sputum. Due to the fact that a more complete collapse of cavities in the

lung is being obtained with the present operation, I believe that an increasing number of patients will be found in this group. The occupations of the patients in Group I are shown in Table III, together with any other data of interest since operation. Table IV shows the number of patients falling into Group I-A, *i.e.*, alive, able to do some work, not symptom free, negative sputum.

TABLE III

PRESENT OCCUPATIONS OF PATIENTS IN GROUP I

Housewife—29	Maid—2	Unemployed—5	Engineer—1
Salesgirl—1	Nurse—1	Elevator operator—1	Clerk—4
Stenographer—1	Technician—1	Houseman—1	Orderly—2
Telephone oper.—1	R. R. attendant—1	Gen'l light labor—3	Bookkeeper—1
	Taxi driver—1	Car checker—1	Merchant—1

Eight of the female patients have married since operation; four of them have had children who are at present 6 months, 4 years, 5 years, and 7 years old.

TABLE IV

GROUP I-A:—ALIVE, NOT SYMPTOM FREE, ABLE TO DO VERY LITTLE WORK, NEGATIVE SPUTUM

No. of Cases	Time Elapsed Since Operation					
	3 yrs.	6 yrs.	7 yrs.	10 yrs.	Total	Percentage
103	1	1	1	1	4	3.9%

One patient has a severe bronchiectasis.
Two patients have marked emphysema.
One patient has moderate cardiac involvement and some emphysema.

The number of patients in Group II, *viz.*, alive, able to do some work, not symptom free, positive sputum, is given in Table V.

TABLE V

GROUP II:—ALIVE, ABLE TO DO SOME WORK, NOT SYMPTOM FREE, POSITIVE SPUTUM

No. of Cases	Years after Operation				Total	Per Cent
	3	5	9	11		
103	2	1	1	1	5	4.8

Occupations: Lithograph artist—1. Housekeeping—4.

In comparison with the figures in Table V are those of Bull's[2] which are 11.6 per cent; of Hedblom's[3] which are 5 per cent and of Hedblom's collected series of 1,235 cases which are 10.9 per cent.

The number of patients that fall into Group III, *i.e.*, alive, unable to work, not symptom free, positive sputum, is shown in Table VI.

TABLE VI

GROUP III:—ALIVE, UNABLE TO WORK, NOT SYMPTOM FREE, POSITIVE SPUTUM

No. of Cases	Years after Operation						—Total	Per Cent
	2½	3	4	5	6	9		
103	1	2	1	3	1	1	9	8.7

The 27 deaths among the 103 patients upon whom this report is based fall naturally into two groups, *i.e.*, the early and the late. The confusion as to what constitutes an operative mortality can be avoided if the deaths are reported with the time interval after operation at which they occurred, since practically all of the deaths within eight weeks are directly due to operation. The large number of early deaths and the fact that they constitute nearly one-half of the total deaths is one of the striking features of all reports. Graham, Singer and Ballon[6] in 1935 reported an operative mortality of 13 per cent in a series of 2,642 collected cases and one of 8.5 per cent in the first four weeks in a series of 140 cases of their own. Hedblom[3] in 1934 in a compilation of 3,811 cases occurring in 24 series, stated that the mortality in the first eight weeks was 10.5 per cent with individual variations from 3 to 21 per cent. His own eight week mortality in 161 patients was 10.5 per cent. Table VII shows the time at which the early deaths in our series occurred with the cause of death in each instance.

TABLE VII

GROUP IV:—PATIENTS THAT HAVE DIED EARLY DEATHS

Of the 27 deaths 9 (8.7% of the total number of patients) occurred within the first six weeks as follows:

2 days	3 days	4 days	7 days	9 days	14 days	35 days
2	1	1	2	1	1	1

Causes of Death

Mediastinal Flutter	Wound Infection	Acute tbc. Pneumonia in Good Lung	Cardiac	Lobar Pneumonia	Pulmonary Hemorrhage	Acute Autotuberculinization
1	1	3	1	1	1	1

Except for more careful selection of cases there is little that can be done to decrease the number of late deaths most of which are due to tuberculosis in some form. It should be possible, however, to lower the mortality which occurs in the first eight weeks. In a combined series of 319 early deaths recently reported,[3] it was found that wound infection was responsible for 8.3 per cent, shock for 8.3 per cent, heart failure for 21.3 per cent, mediastinal flutter for 2.3 per cent and pulmonary complications for 38 per cent. The number of deaths occurring from shock can certainly be decreased by an operative technic that demands careful hemostasis and lack of trauma; and by dividing the operation into many stages, no one of which is of enough magnitude to cause a dangerous degree of shock. No patient in our series died from shock following operation. The resection of not more than three ribs at a single stage will do much to prevent shock and abolish mediastinal flutter.

I am in accord with Graham[6] in believing that there are relatively few true cardiac deaths following thoracoplasty and that many patients whose deaths are ascribed to heart failure really die of autotuberclinization and acute

tuberculous pneumonia of the good lung. Allen[7] believes that the anoxemia due to an insufficient vital capacity remaining after collapsing one lung is frequently confused with heart failure—a feeling which I likewise share. A careful preoperative study of the lung volume as advocated by McIntosh[8] after the method of Christie,[9] is a definite aid in determining the reserve upon which a patient can rely after operation. The highest percentage of early deaths is due to pulmonary complications, the chief one of which is an acute tuberculous pneumonia involving the good side. Undoubtedly some of these follow a reactivation of a supposedly healed focus in the good lung. The added "strain" thrown upon the sound lung was commonly thought to be responsible for this but Churchill[10] feels that this factor may be greatly discounted. He has shown that if a harmful strain is ever thrown on the collateral lung, it is most likely through the increase in the rate or depth of breathing which results from the increased carbon dioxide content of the blood entering the general circulation from the collapsed, but actively circulated lung. A large percentage of the cases of acute tuberculous pneumonia in the good lung is due to the aspiration into it of the contents of cavities which are compressed when the diseased lung is collapsed. The mechanism by which this occurs has been well described by McCordock and Ballon.[11] They state that the development of acute tuberculous pneumonia in the good lung in many cases depends upon the amount of compression obtained and upon the location of such compression. It is the squeezing out into the bronchial tree of the infected contents of tuberculous cavities and the aspiration of such contents into the already sensitized good lung that produced the tuberculous pneumonia. The experimental work of Rich and McCordock[12] has shown that such a mechanism does exist and that by it massive tuberculous pneumonia may be produced within a few days. The latter authors also have shown that the severity of the pneumonia is dependent upon the number of bacilli which are aspirated into the good lung—a fact that explains the recovery of some patients and the rapid death of others. A considerable amount of edema accompanies this type of pneumonia and on this account many cases are unrecognized and death is attributed to edema of the lungs. In this connection the experience of Allen[7] is interesting. In one of the hospitals in which he operated, he had not for seven years had a single instance of acute tuberculous pneumonia following a thoracoplasty operation. He ascribed this to the fact that he had to operate at that particular hospital only in the afternoon and by that hour the patients had cleaned their lungs during their morning coughing even better than they could have by postural drainage. It is largely for fear of aspiration during operation that I have performed all of my operations under local anesthesia. Hedblom quotes Denk[3] as believing that postoperative mortality is influenced by the season of the year at which the operation is performed and states that between October and March his mortality was 13.8 per cent in 165 operations, whereas between April and September, it was only 4.5 per cent. Hedblom's[3] own experience, however, was just the opposite with a mortality of 2.1 per

cent for winter and 6.2 per cent during the summer. Our series shows 4.3 per cent from October to March and 4.4 per cent from April to September.

Apparently there is little uniformity of opinion as to the relationship between sex and mortality rate or between the side operated upon and the early mortality. This series shows a higher mortality rate for females (9.5 per cent) than for males (7.5 per cent) and a higher rate (10.1 per cent) for left-sided than for right-sided operations (9 per cent); a higher rate for the left side in males (5 per cent as compared to 2.5 per cent for the right side); and for the left side also in females (6.2 per cent as against 3.1 per cent for the right side).

TABLE VIII

GROUP IV:—PATIENTS THAT HAVE DIED LATE DEATHS

Of the 27 deaths 18 (17.4% of the total number of patients) occurred after 3 months following operation as follows:

3 mos.	4 mos.	1 yr.	1½ yrs.	2 yrs.	3 yrs.	3½ yrs.	4½ yrs.	6½ yrs.	8 yrs.	9½ yrs.
1	3	3	1	1	3	1	2	1	1	1

Causes of Death

Tuberculosis of the Good Lung	Generalized Tuberculosis	Peritonitis Following a Ruptured Cecal Ulcer	Cardiac	Cerebral Hemorrhage	Mediastinal Lymphosarcoma
9	5	1	1	1	1

It will be noted in Table VIII that 83.3 per cent of the late deaths were due to tuberculosis in some form, usually more often a spread of the disease to the good lung. This has been the experience of other authors. Three of the late deaths in this study were due to causes unrelated to operation or to the disease and occurred in patients who were alive, with negative sputum and working nine and one-half, three, and three and one-half years after operation.

SUMMARY

(1) A series of 103 cases of pulmonary tuberculosis which were treated by thoracoplasty is reported.

(2) At least two and one-half years have elapsed since operation in every case and as much as 11 years in some of them.

(3) Of the 103 patients—58 are working and have negative sputum; four others have negative sputum, but are unable to work; five are able to do some work but still have a positive sputum; nine are unable to do any work and have positive sputum; and 27 are dead.

(4) The causes of the deaths have been analyzed. Emphasis is laid on the large number of early deaths following operation.

(5) Nearly all of the late deaths were due to tuberculosis in some form.

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THE USE OF INTERCOSTAL MUSCLE IN THE CLOSURE OF BRONCHIAL FISTULAE

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THE large majority of bronchial fistulae uncomplicated by persistent pulmonary disease heal spontaneously or can be cured by relatively simple surgical measures. Some, however, do not respond readily to the common methods of treatment. Among these, in our experience, are the fistulae that follow drainage of central abscesses of the lung. In many such cases the terminal condition is as illustrated in Figures 1 and 2. A more or less cylindrical channel about one-half inch across and one to two inches in depth represents the line of drainage. The walls of this channel are widely separated during inspiration and closely apposed on expiration, the result of this constant movement being to retard healing indefinitely. The operative procedures required for the opening of the original abscess will have usually destroyed so much of the adjacent tissue that grafts from the superficial thoracic muscles cannot be found for closure of the fistula. In the treatment of several of these cases we have successfully used, as plastic material, intercostal muscle, not infrequently taken from a space a considerable distance from the fistulous opening.

The intercostal muscle possesses almost every characteristic desirable in a muscle flap: it is easily separated from the surrounding tissues; it retains intact its vascular and nerve supply and has a mobility rarely obtained in any other muscle that can be used for plastic work, and that without impairment of its circulation.

Operative Procedure.—The scar of the original wound is excised and one or both margins are widely enough retracted to expose the nearest intercostal muscle uninjured by the primary operation. While the muscle can be dissected from its bed without resection of the adjacent ribs, in practice it is better to remove these superiosteally first. The operation can then be carried out more rapidly and with less danger of injuring the blood supply of the proposed transplant. The structure and strength of the thoracic cage is very little if at all affected by the more extensive procedure. The ribs having been resected for a sufficient distance to provide a proper length of graft, the muscle is cut across anteriorly and stripped from the underlying pleura (Fig. 3). The free end is then implanted into the depth of the fistulous channel and held in position by a few interrupted catgut sutures (Fig. 4). The wound is then closed, a small tube only being left for drainage. Except for a slight, blood tinged expectoration lasting a few days, there have been no complications. Healing has taken place promptly in four of the five cases operated upon. In

one patient an unrecognized bronchiectasis was the presumable cause of failure.

Though it is somewhat outside the scope of this paper, we have also used a modification of this procedure as a primary method of treatment in a case

FIG. 1.—Persistent bronchial fistula following drainage of abscess of the lower lobe.

FIG. 2.—Enlargement of Figure 1 illustrating cylindrical channel leading to the fistulous openings.

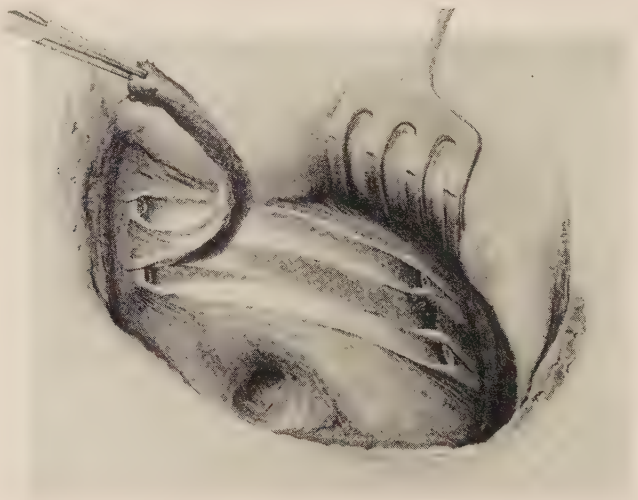

FIG. 3.—Preparation of the intercostal muscle graft.

of chronic abscess of the lung. The abscess, of six months' duration, lay in the midaxillary line immediately beneath the seventh and eighth ribs on the right side. As viewed under the fluoroscope its shadow did not move with respiration and it was apparently fixed to the chest wall over a considerable

area. Through a lateral oblique incision the seventh and eighth ribs were resected subperiosteally for a distance of six inches. The intercostal muscle was then separated from the pleura and retracted upward to permit a visual and digital examination of the latter structure and to determine its relationship to the underlying lung. As the pleura was obviously obliterated, the wound was thoroughly "bipped" and the abscess opened. The cavity was somewhat smaller than a baseball in size, contained a thick purulent exudate and presented a smooth lining in which were visible the openings of several fistulae. It was felt that convalescence might be shortened by partially filling

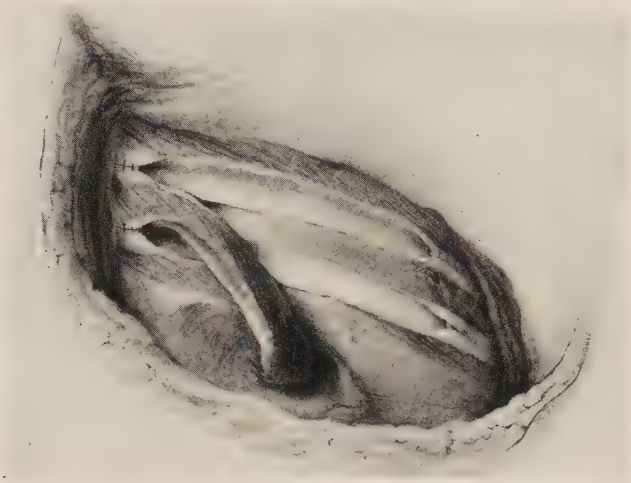

FIG. 4.—Implantation of the intercostal muscle into the depth of the fistulous tract.

the cavity with the intercostal muscle. This was done much as in the cases cited above, the intercostal muscle being curled within the hollow and held in place by a few sutures and some light gauze packing alongside of which a tube was placed as an additional safeguard. Otherwise the wound was closed. Healing took place very rapidly and so far as we could tell no air escaped through the wound at any time. The incision was completely closed in five weeks and the patient has remained well.

DISCUSSION OF THE PAPERS OF DOCTORS SPEED, ALEXANDER, CARTER AND SHENSTONE

DR. HOWARD LILIENTHAL (New York, N. Y.).—You have had the privilege of listening to unusual and in some instances epoch marking papers. I shall confine my remarks particularly to those on the surgical treatment of tuberculosis. Doctor Carter is to be complimented on his pronouncement, that I have always believed in and have ventured to publish, that the surgeon has a moral obligation to patients with tuberculosis who appear doomed unless something miraculous happens. It will happen if enough of these patients are treated surgically. The only thing that would prevent my operating in a case of this sort, or almost the only thing, is that there should be sufficient functioning lung to sustain life in a fairly normal manner. Otherwise, I do not care whether they have laryngeal, peritoneal, or bone tuberculosis—and I have had them all three in one patient, for that matter. If there is enough

functioning lung to keep the patient alive he ought to be given a chance through surgery, if the case is technically operable, and success will follow in enough instances to prove the truth of this principle.

There is one thing that I have noticed which has not been presented, and I have not seen it mentioned elsewhere. My attention was called to it by Doctor Amberson in New York in a case in which a patient died following an upper stage thoracoplasty from infection of the opposite lung, in which every possible effort was made to prevent a spill-over at the operation, which I am quite certain did not occur at that time. He developed a peculiar cough, with a mobile mediastinum, which Doctor Amberson described as a *paradoxic cough*, meaning that during the period just before the expulsive part of the cough, when the larynx was still closed, there was, literally, a cough into the opposite lung. As a matter of fact, postmortem examination also showed that the opposite lung was actively diseased.

Permit me to mention very briefly a paper that I have sent to the Journal of Thoracic Surgery, in which I describe a new form of an apicolytic thoracoplasty. I am convinced that in apicolytic thoracoplasty, with or without further thoracoplasty, the first rib does not need to be cut. I have had experience in six cases with this procedure, but that is sufficient to show that the immediate result in cases of this sort may be good. If the extrapleural space, that you have secured, is packed with rubber dam and left in place for four or five days, and then drained from below, the lower surface of the first rib acts as a perfect buffer for pushing the infected apex down, with obliteration of the pulmonary cavity. One advantage in saving the first rib in this way, and without prejudice to the operation otherwise, is that there is no danger of injuring the large neighboring vessels and nerve plexus during the manipulation, and that, therefore, the neuritis which so frequently follows, will not occur. That is entirely done away with, and it is an advantage well worth considering.

Doctor Alexander's paper was excellent. Most of the things that he does are, and I have greatly enjoyed hearing him. He speaks of operating in front of the original incision, when secondary operation is necessary to take away more rib. I used to do that, but I do not do it any more, and I doubt that it is ever necessary. At least it has not been, so far, in my cases. Through the original incision, the chest having been already pretty well collapsed, you can, with little trouble and with almost no hemorrhage, work around to the cartilages and take away as much rib as may be necessary. I do not believe that Doctor Alexander will continue operating through another incision when he has once given this a fair trial.

As to the formalin method of preventing the formation of rigid bone, it seems to me that I would require a successful demonstration of its harmlessness before I would want to employ it. As it is, when you take away large sections of rib, you are quite apt to get a mobile part of the chest wall, which I think in tuberculosis is a great disadvantage. The desideratum is *rigidity* in the new position of the chest wall.

For the last two years I have used electric surgery for the skin incision, and electrocoagulation for taking care of the vessels, except those that spurt, notwithstanding its application; these are tied. In a secondary operation, when I had not taken enough of the ten ribs away at the first procedure, I was able to perform the entire operation and take out more of all the ten ribs, almost to the cartilages, without using a single piece of catgut. Perfect primary union followed. This method will not only save a lot of time but will be extremely valuable. It cannot be used, by the way, when ethylene or cyclopropane is being employed, for obvious reasons. Also the incision

through the skin has to be made quickly and there must be a current that will deliver the kind of cutting electricity which you may use at high speed. Otherwise there is danger, of course, of slow healing.

There is one question I want to ask Doctor Shenstone about the abscess that was so perfectly cured by the intercostal muscle flap. Was that a putrid abscess?

Dr. Martin Buel Tinker (Ithaca, New York).—There are three things I should like to bring out in connection with these interesting papers.

In the first place, Doctor Lilienthal has just mentioned the use of electrocoagulation in this work. Those of you who have used it know how much time it saves. You realize, too, that with the electric cutting current you get sealing of the lymphatics and blood vessels which prevents infection of your wound. Those are two decided advantages. And the third is that you do not leave foreign material in the wound. Doctor Lilienthal in another discussion called attention to the fact that in operating upon the chest the current should not be used if you are working in the vicinity of the heart.

Doctor Speed's report of the case of tumor of the chest wall associated with tumor of the liver is, I believe, the only tumor of the character on record. It was a very formidable procedure and required a lot of courage to go into the peritoneal and chest cavities as extensively as he did, in order to remove the large liver tumor in connection with that of the chest wall. He failed to say that this woman not only lived but has borne two children.

The third point I should like to bring out in connection with Doctor Speed's paper is the frequency of tumors of flat bones, and particularly of the chest, in connection with tumors of the thyroid. He spoke of adenocarcinoma. You are all familiar with the paper of Cohnheimm, published nearly 50 years ago, in which he called attention to the fact that certain tumors which clinically and pathologically are apparently benign, metastasize to the flat bones. Some French observers believe that if we search far enough we find actual malignancy, but I think there are some 75 instances of recurrence of this kind in which the tumor apparently was perfectly benign.

Dr. Casper F. Hegner (Denver, Colo.).—Doctor Alexander covered the subject and left nothing for discussion. We all know of the work he has done in the surgery of tuberculosis.

The character of the lesion within the lung, or within the pleura, the extent of the lesion as well as its location, determine the length of the segments of the ribs to be removed. I believe, as to the lateral and the anterior costectomy, if sufficient length of rib is removed with the transverse processes, these operations will be unnecessary. Secondary operations, of course, can be approached through the posterior incision. No matter how many operations have been performed, it will rarely be necessary to employ other than the posterior approach. I have not found difficulty solely with the size of the cavity. Of course we secure the necessary relaxation in order to permit nature to cause fibrosis of this cavity. More difficulty has been experienced with the character of the wall of the cavity, and the nature of the surrounding pericavitary zone. Sufficient deribbing must be effected in order to secure a degree of relaxation sufficient to permit nature to initiate fibrosis.

We also had a little difficulty in the number of ribs removed. If we remove the seventh rib we have to take the eighth in order to eliminate the snapping of the scapula, which causes a great deal of discomfort.

In regard to the application of formalin, we have used that a good many times. It does not entirely prevent regeneration of the ribs, but it does retard

it sufficiently to permit the interval between stages to be very much more prolonged.

Tumors of the chest wall, referred to by Doctor Speed, have been interesting. We have had three cases of this myxofibrosarcomatous condition of the chest. One was in a man of 70, in whom two or three operations had been performed, but it has recurred. I think the multiplicity or complexity of the cytologic examination is very much more important than a negative report from the pathologist. Whenever you have a multiplicity of cellular elements, you know it is either malignant or will become so.

Dr. Stuart W. Harrington (Rochester, Minn.).—Tumors of the chest wall often present greater surgical problems than tumors that are entirely within the thoracic cage, because surgical removal of those involving the thoracic wall and projecting into the thoracic cavity are associated with all the dangers of a transpleural operation as well as necessitating reconstruction of the thoracic cage, which should be completely closed. Doctor Speed pointed out that the majority of these tumors are malignant, which is of great surgical importance because they demand radical removal, which often involves large areas of the chest wall.

My experience consists of 39 cases in 30 of which the tumors were malignant and in nine, benign. Benign tumors are much more satisfactory to treat, for they are cured by complete removal of the tumor, and surgical removal is usually not associated with extensive removal of the chest wall and does not require such extensive reconstruction of the thoracic cage. Cases of malignant tumor are not so gratifying from a surgical standpoint, for they not only present greater operative risk but the disease is usually so extensive at the time of operation that complete eradication of it is rarely possible. The average length of life in the 30 cases in which operation was performed has been two years and five months. Discouraging as this is from the standpoint of results, some patients apparently are cured. One patient of this series who presented an osteosarcoma which originated from the rib and projected into the thoracic cavity, is now living without evidence of recurrence 11 years after radical operative removal of the growth.

I think the surgical problem depends a great deal on the situation of the tumor. It might be said that the more favorable tumors are those which are situated posteriorly and which do not involve the spine. The surgical difficulties increase when they are situated anteriorly in the chest wall; this is attributable to the increased difficulty of complete closure of the thoracic cage after removal of the growth. Complete closure of the thoracic cage is more easily accomplished following removal of the posterior tumors because the muscles of the posterior chest wall can be utilized in the closure of the thoracic cavity. As you go toward the sternum and diaphragm, there is increased difficulty in reconstructing the thoracic cage.

I shall present three cases very briefly.

Case 1.—The first case is that of a benign chondro-osteoma originating in the chest wall and extending into both anterior and posterior thoracic cavities. The difference in the roentgenologic shadow of this tumor in the various roentgenograms demonstrated the value of multiple roentgenograms, particularly of lateral as well as of anteroposterior views, in all cases of intrathoracic tumor. The lateral views in this case showed the tumor to project well into the posterior thorax. It was removed through a posterolateral incision. The anterior portion of the tumor was removed transpleurally through this incision, without incising the skin of the anterior chest wall. This permitted complete closure of the defect, posteriorly, with the scapula and muscles of the posterior thoracic wall. The patient obtained a very satisfactory result and now more than four years have elapsed since operation. He has a flaccid anterior chest wall in the area where

the tumor was removed which is troublesome only when a cold develops; then it is necessary for him to support the chest wall when coughing.

Case 2.—The second case is that of a large, recurring malignant endothelioma of the lower chest wall involving the diaphragm. Surgical removal of this tumor required very extensive removal of the lateral chest wall, including about one-fourth of the diaphragm. The opening in the abdominal cavity was closed by suturing the paralyzed diaphragm, after interruption of the phrenic nerve, to the chest wall above the area of the chest wall. The defect in the chest wall was closed with the posterior muscles. This patient lived two years and three months after the operation and died of recurrence.

Case 3.—The third case is that of a malignant tumor in the posterolateral chest wall. The growth involved the rib and proved to be an osteogenic sarcoma of moderate size. The growth was removed in two stages. The first stage, directed to walling off the pleural cavity, failed, as a pleural effusion developed, necessitating immediate transpleural removal of the growth. The man made a satisfactory recovery and is now living, 11 years after operation, with no signs of recurrence. This case again exemplifies what Doctor Speed has emphasized in his presentation, namely, that the most important consideration relative to malignant growths of the chest wall is their early radical removal.

Dr. Owen H. Wangensteen (Minneapolis, Minn.).—About 20 years ago, Doctor Kanavel of Chicago pointed out the great virtue of pedicled muscle flaps in the closure of cavities of diverse natures. Those of us who have employed this expedient for such purposes can attest its worth. It was Abrasanhoff, a Russian surgeon, I believe, who first employed a pedicled muscle flap to secure closure of a bronchopleural fistula. He first sutured it over the bronchial stoma and later recommended implanting a slip of muscle into the fistulous tract in the lung in suitable cases. Doctor Pool has also, together with one of his associates, Doctor Garlock, extolled the merits of this method.

It is, to be sure, only the persistent bronchopleural fistula in which attempts at operative closure of this abnormal communication are indicated. The ordinary bronchopleural fistula which contributes to the continuance of empyema and the development of chronic empyema, usually closes spontaneously with the establishment of adequate and dependent drainage of the residual empyema cavity. In the presence of a suppurative process in the lung, which is still active, a bronchopleural fistula serves a useful purpose and efforts directed to obliterate it are not indicated.

That viable muscle tissue resists digestion is well illustrated in its survival in a chronic duodenal fistula, into which I implanted such a pedicled muscle flap some years ago. This fistula had apparently followed a nephrectomy and had remained refractory to all the usual methods of closing such a fistulous opening. Success attended implantation of a pedicled muscle flap.

The bronchial fistulae which, in particular, have concerned me are those coexistent with a large empyema cavity. I have had an interesting experience with two such cases in which multiple stomata were present. In each instance, after the prolonged establishment of adequate drainage, a pedicled muscle flap was mobilized and sutured over the stomata. Implantation was impractical because of the number of openings. The improvement in each case was startling; the pleurocutaneous wounds closed entirely and the patients remained afebrile. In each instance, after the elapse of some months, the patients became febrile again, owing to the accumulation of fluid in the persistent pleural cavity. A number of years ago (1920) Doctor Heuer reported attempts at sterilizing empyema cavities and closing them without obliterating them. The early favorable issue attending closure of the bronchial stomata in the two instances referred to above lent temporary confirmation to Heuer's suggestion that this objective might be accomplished. In both instances,

however, subsequent drainage and obliteration of the residual empyema cavity was found necessary.

The use of intercostal muscle as suggested by Doctor Shenstone for implantation into a broncho-pleural fistulous tract would appear to be a very simple and readily available expedient. I should like to refer in this connection to another use of the intercostal muscle bundle which I have practiced in the treatment of empyema. In a large empyema cavity with rigid walls, it is frequently found necessary to sacrifice, after adequate decostalization, the soft tissues of the chest wall, that is, the parietal pleura and the intercostal muscle bundles together with their accompanying blood vessels and nerves, in order to obliterate the empyema cavity. Such a procedure, unfortunately entails section of the nerves to the chest wall, and when sacrifice of the lower intercostal nerves, particularly from the sixth to the ninth or tenth, is made, a large abdominal hernia is likely to develop. In 1932, when operating upon such an empyema, it occurred to me that the intercostal nerves might be preserved by a process of ribboning, in which, after excision of ribs, long horizontal incisions were made in the avascular rib beds, converting the intercostal muscle bundles and thickened parietal pleura into a series of parallel ribbons. By excising wedges from the thickened parietal pleura at either extremity of the incision, these ribbons were made to fall in and were used at the same time as a fill-in substance to help obliterate the empyema cavity. To date, this procedure has been used in three instances with large chronic empyema cavities, with satisfactory results, and abdominal hernia has been averted. The details of the operative technic of this procedure were described in the Journal of Thoracic Surgery, **5**, 27, October, 1935.

Dr. Evarts A. Graham (St. Louis, Mo.).—I should like to speak briefly first about the subject of bronchial fistulae.

Doctor Bettmann of Chicago, some ten or 12 years ago, called attention to the fact that the normal mechanism of healing of a bronchial fistula is by circular contraction. Bronchial fistulae will, in my experience, always heal if they have a chance to contract in a circular direction. If they do not have that opportunity, they will not heal. Consequently, in planning an attack on a bronchial fistula, we have, it seems to me, one or two ideas which we can utilize. We can either mobilize the lung so that circular contraction may occur, or we may plug the bronchus with something so that circular contraction will not be necessary.

The use of muscle laid against the opening of a bronchial fistula, is an effort to make it close, is an old procedure, but one which, at least in my experience, has often failed. Doctor Pool, however, a few years ago, called attention to the great advantage in using muscle, and plugging it into the bronchus. Now you can see, in the light of what I have just said about circular contraction, the great virtue of this principle of plugging the bronchial opening rather than laying muscle against it, because if the bronchial opening is plugged with the muscle flap, the lumen is completely filled with the growing tissue which is put in there, circular contraction is no longer necessary, and, to make a long story short, the closing of the fistula is accomplished. That is a very different matter from merely laying muscle, even if it is a flap of viable muscle, over a bronchial opening.

I think Doctor Wangensteen's suggestion in regard to chronic empyema is an excellent one, having very distinct merits, and again it serves, somewhat, to accomplish the closure of bronchial fistulae by a certain amount of mobilization of the lung which permits circular contraction.

I want to say a word or two also about Doctor Speed's paper on tumors of the chest wall. I think it is very essential in a paper of this sort before

a group of general surgeons, to mention that there is very great, immediate danger present in operating upon these tumors of the chest wall. Certainly no one should undertake an operation upon one of these tumors without being prepared to use intratracheal anesthesia, if necessary, in order to save the patient. It is quite true that in some cases one can remove the entire side of the chest wall without any special precautions to maintain the intrathoracic pressure, but those instances are great exceptions.

Again, there is another feature about these tumors that I should like to mention as a warning, and that is that one can never be quite certain from the roentgenologic appearance about how large the tumor actually is. Some of these tumors, which contain a minimal amount of cartilage and bone and more or less soft tissue, may have such tremendous extensions inside the chest wall that one is amazed when the chest is opened to find that the tumor is considerably larger than he had anticipated before undertaking the operation. For example, at the present moment I have a patient who had a tumor of undiagnosed nature before I operated upon him, a tumor which cast only a very faint shadow. To my amazement, when I opened into the chest I found a large lipoma of the pleura, projecting into the pleural cavity. This is a very rare tumor, by the way. It was necessary to make an exceedingly wide opening in the chest wall and to remove a very considerable amount of parietal pleura with the tumor, as it was so firmly attached to the pleura that it could not be separated. It would have been an easy mistake to have concluded, before the operation, that the removal of the tumor would be simple. Such was certainly not the case.

Again, there are other tumors to which reference should be made, namely, the group of neurofibromata, von Recklinghausen's disease, which sometimes occur along along the intercostal nerve, but yet may be connected with portions of the tumor, which may arise really within the vertebral column; tumors of the type which Doctor Heuer and Dr. Andrus discussed a few years ago under the name of hour-glass tumors. I have a patient with such a tumor in the hospital now, a young woman with an enormous intercostal neurofibroma, which involved the entire intercostal nerve. There was no indication from the external appearance, or from the roentgenographic appearance either, for that matter, of the enormous extent of this tumor.

Dr. Arnold Schwyzer (St. Paul, Minn.).—Showed several lantern slides of a case of an endothelioma of the pleura upon whom he had operated, as complementary to Doctor Speed's presentation, and cited the appended case report.

Case Report.—Male, aged 23, student. In January, 1929, he began to have pain in the lower right side of his chest anteriorly. A roentgenogram showed a lobulated mass with a pedicle attached to the right pleura. It was thought to be either an encapsulated serous pleurisy or a cyst. It was tapped and 10 cc. of a straw-colored fluid were removed which proved to be negative when cultured. The patient then felt somewhat better.

On May 9 and June 15, 1929, roentgenograms were taken but showed no change in the shadow. He then went away and was not seen until November, 1929, at which time he complained of a swelling over the right lower chest and also severe pain. The roentgenogram taken at this time showed that the mass had grown considerably and the diagnosis was then made of probable endothelioma of the pleura at the right base. Tapping yielded 5 cc. of fluid. A tuberculin test was negative. On December 22, 1929, a biopsy established the diagnosis of endothelioma. It did not seem very malignant according to the miscroscopic picture. The case was referred to me January, 1930.

Though endothelioma of the pleura is usually a far spread condition, we had here an

apparently localized tumor. If nothing were done there would be a long period of desperate hopelessness before the patient, with Death steadily stalking closer toward him. The tumor was so clearly outlined toward the lung, though reaching far inward, that we thought possibly it had not invaded it, though the attachment to the diaphragm made this doubtful. We figured that in case the tumor had no attachment the outlook was quite reassuring, but if it were firmly adherent to or had grown into the lung, a clean resection of the adjoining lung was possible. The degree of extension into the diaphragm was uncertain, but with a wide and sufficient opening we could perhaps cleanly resect this area. There was nothing discovered elsewhere in the body to indicate that this tumor was perhaps secondary, as tumors of the pleura often are. There had been no previous operations for apparently minor skin or other growths. The pleura, apart from the tumor, seemed entirely normal.

A very simple overpressure apparatus was constructed by attaching two intranasal tubes to an oxygen tank with a tapping sidewise for control of the pressure. This consisted of a rubber tube from which a glass tube entered for 20 cm. into a deep basin filled with water. Thus the pressure was regulated.[1]

Operation.—January 8, 1930. After an hypodermic of 1/6 gr. of morphine and 1/150 gr. of atropine, 2 cc. of spinocaine was injected into the spinal canal. We use a little modification of Pitkin's method in that we turn the patient onto the stomach immediately after the injection and make use of the dorsal curve of the spine to allow the solution to rise to the desired height. This works admirably and has been adopted by several of my St. Joseph's Hospital colleagues. The anesthesia was perfect and the patient in good condition throughout the operation apart from some transient nausea. An incision, about 14 cm. long, was made parallel with the ribs, and included the scar of the biopsy incision. The sixth rib was removed for a length of about 12 cm., and the mass could now be better appreciated. It seemed rather intimately connected with, and directly under, the seventh rib. The seventh and eighth ribs were divided anteriorly and posteriorly and allowed to remain attached to the growth. A small opening in the pleura allowed air to enter slowly. Overpressure was installed from an oxygen tank. The respiration was very quiet, though the lung was allowed to become considerably collapsed. The patient's color was very good and remained so. The usual coughing spell on interfering with the pleura did not occur. The pleura was then widely opened; the lung was nowhere adherent and its surface as well as the parietal pleura was perfectly normal. On lifting the rib with the tumor the attachment to the diaphragm tore loose. All the intercostal structures including the pleura from the sixth to the eighth rib were removed in one piece. There was only a very superficial involvement of the diaphragm, and an area about 3 cm. in diameter, near the periphery of the diaphragm anteriorly, was excised and the defect closed with catgut. Closure of the wound was effected by suture of the extracostal muscles with continuous catgut. Before pulling the last suture through, the patient was requested to strain and thus empty his pneumathorax. Air tight continuous skin sutures of linen were introduced.

Recovery was smooth. Patient was out of bed four days after the operation, feeling well. By February 17 he had gained ten pounds. The postoperative exudate absorbed rapidly.

Radium was applied to the chest in considerable quantity. Up to the end of 1930 he had had about 13,000 milligram hours, filtered by 1 Mm. of lead and 12 Mm. of wood.

On October 2, 1930, the entry was made, "Patient looks very well, weighing more than ever before; says he feels perfect." Roentgenologic examination showed a normal lung field on the operated side, but a haziness in the left lung field suggestive of some thickening of the pleura.

On January 8, 1931 (one year to the day after the operation), the patient suddenly felt a sharp pain in the left side which persisted several days. A similar attack occurred in February and a third in March, at which time he returned to the hospital. An involvement of the left side was apparent. By June dyspnea became severe and the whole

left side was dull. Two attempts were made to obtain fluid, with negative results, though the left side bulged markedly. A nodule in the left supraclavicular space was then noticed which had grown to the size of a lemon at the time of death, August 26, 1931.

Autopsy.—(University Department of Pathology.) The right pleura and the right diaphragm were found to be normal. There was no evidence of any involvement of the right diaphragm. At the bottom of the right lower lobe there was a small nodule and another on its anterior surface. No pleural adhesions were noted. The left pleura was intimately adherent to the lung and diaphragm. The left lung consisted entirely of white rounded tumors varying in size from three to eight centimeters in diameter. The mediastinum did not appear invaded. Metastases had occurred to the ligamentum teres hepatis and to the periaortic and cervical lymph nodes. Microscopically the lung showed endothelioma.

Neither Sauerbruch nor Lilienthal report an operation for endothelioma of the pleura in their books on Surgery of the Chest. Endotheliomata which hold a position midway between connective tissue tumors and carcinomata have important clinical characteristics. They differ from sarcomata and carcinomata by their slower growth and by the fact that they usually do not produce metastases until late, when the tumor has reached a considerable size. The neighboring lymph channels are not invaded early, as is usual in carcinoma. The prognosis is better, therefore, after thorough removal. However, they are known to be exceptionally prone to recur locally, much more so and earlier than carcinomata.

Whether in this instance the two aspirations and the biopsy had an influence on the spreading of the neoplasm is problematical. It is, however, of interest that even one year after the appearance of the first symptoms the operation furnished a local cure.

REFERENCE

[1] Schwyzer, Arnold: Notes of Surgery of the Mediastinum. ANNALS OF SURGERY, **75**, 53.

DR. EUGENE H. POOL (New York, N. Y.).—In connection with Doctor Graham's remarks, it is true that a few years ago, with Doctor Garlock, I reported a method of a pedicle muscle flap for the closure of a bronchial fistula. Subsequently we found that this method had already been published in a foreign journal, so we can lay no claim to originality. It is, however, an extremely satisfactory one and has been used in an appreciable number of cases with success.

DR. JOHN ALEXANDER (Ann Arbor, Michigan) closing.—Doctor Lilienthal brings up a point he made a number of years ago, that in performing a secondary thoracoplasty—that is, revising a thoracoplasty which had been badly performed before—it is perfectly possible to resect the anterior portions of the ribs by reopening the posterior incision, resecting the regenerated ribs posteriorly and carrying the resections much farther forward than they were carried previously. The point I wish especially to make is not in regard to a secondary thoracoplasty but in regard to the horizontal staging of the primary thoracoplasty.

By this I mean that only exceptionally should the resection of the ribs be carried as far forward as their costochondral junctions during a posterior thoracoplasty operation. If additional collapse should be needed the anterior portions of the ribs and the cartilages may be dealt with in a number of ways, thereby horizontally staging the costal resections and preventing too sudden pulmonary collapse and undue paradoxical respiratory motion.

Since permanent anterolateral mobility may prove to be harmful to the tuberculous lesions, as well as to respiratory functions, the periosteum of the

ribs that have been removed should be rubbed with 10 per cent formalin only from the vertebrae to approximately the posterior axillary line, where the rigid scapula prevents undue movement of the decostalized thoracic wall. In cases in which the pleura is relatively rigid and in which it is important that prolonged contraction of the diseased lung, or an empyema cavity, should not be prevented by costal regeneration, all the periosteum of the resected ribs should be formalinized.

After having used electrocoagulation of vessels for approximately two years I abandoned it because occasionally a reactionary hemorrhage and hematoma occurred in spite of the wound having been left perfectly dry at the conclusion of the operation.

In removal of large tumors of the thoracic wall I should like to suggest the use of the Drinker Respirator in cases in which great postoperative paradoxical movement results in anoxemia and impending cardiorespiratory decompensation. The Respirator can be regulated so as to compel the decostalized portion of the thoracic wall to move outward during inspiration, instead of inward as in paradoxical respiration. Some years ago, before the Respirator was available, I lost a tumor patient from paradoxical respiration who, I believe, would have been saved by its use. Several years ago a patient, in whom death was imminent from great paradoxical movement following the third stage of a thoracoplasty, was dramatically saved by being placed in the Respirator. As I recall, the pulse rate dropped from 160 to 110 within 15 minutes. Since cardiorespiratory compensation gradually developed within a day or two, the patient needed to be in the machine only intermittently for several additional days.

Dr. Kellogg Speed (Chicago, Ill.) closing.—In response to Doctor Tinker's prompting, may I say that the patient with the large tumor of the chest wall, with the resection of the abdominal wall, the liver and the diaphragm, has since borne four children. She is robust and weighs 210 pounds at the present time. She has recently had an hysterectomy and assures me that there will be no more children.

Doctor Harrington's illustrations show the technical difficulties of removing some of these tumors in their advanced stage. When they become myxomatous and infiltrating, they may invade the pleura or the lung to a certain extent, and it is not easy to lift them away from the lung. It is somewhat of a comfort if, on opening the chest, one sees the lung collapse away, following which radical excision may be performed; but if the pleura is infiltrated and the lung is adherent, the procedure may not be possible, that is, the decortication and removal of the surface may not be possible at that one sitting. Doctor Schwyzer's case is perhaps another instance showing the necessity of being sure about primary and secondary tumors. For that reason, I should like to stress again my remark that any tumor of the thoracic wall warrants removal, or a very thorough investigation.

Dr. Norman S. Shenstone (Toronto, Canada) closing.—In regard to Doctor Lilienthal's question, this was a septic abscess containing all of the various putrefactive organisms found in the mouth, and with foul expectoration. It was, however, a chronic abscess and therefore had a smooth wall.

I had not seen the use of intercostal muscle taken some distance from the fistula itself described before, but make no claim of originality in using this procedure. In a very large proportion of cases that I have seen with fistulae of this sort, the ordinary muscles of the chest wall have been so widely destroyed that it has been quite impossible to obtain muscle tissue from the usual superficial sources.

TRAUMATIC SURGERY OF THE LUNGS AND PLEURA

ANALYSIS OF 1,009 CASES OF PENETRATING WOUNDS

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DURING the 14 years from 1922 to 1935 there were admitted to the Emory University Division (for Negroes) of the Grady Hospital of Atlanta (municipal) 1,187 patients suffering from wounds of the chest. Of these injuries 1,009 (85 per cent) were diagnosed as penetrating the lungs or pleura, and 162 (15 per cent) were diagnosed as nonpenetrating. In addition, 16 cases of stab wound of the heart and one case of stab wound of the pericardium were sutured, with a recovery of 50 per cent. The possibility of cardiac damage should be considered in every patient with thoracic trauma.[1] Many of the fatal injuries of the lungs and pleura also included wounds of the heart and great blood vessels which were not diagnosed or treated. The proportion of male to female patients was three to one, the average age being 27.

The majority of these patients apparently had small wounds of the lungs or pleura caused by slender, sharp pointed weapons such as an ice-pick or "swish-blade," four inches long, so called because the assailant "swishes" the knife open to stab his victim. Care was exercised in determining which of these cases were penetrating and which were nonpenetrating injuries, although in a considerable number it was difficult, if not impossible, to tell the difference. This especially was true when the clinical and roentgenologic findings did not agree, as in the following case:

Case 1.—A Negro boy was stabbed in the right fifth intercostal space, anterior axillary line, and was brought immediately to the hospital. A definite sucking wound was present, and was sutured. The patient had some pain and dyspnea. On admission the pulse, temperature and blood count were normal, but 24 hours later the pulse rose to 120, and temperature to 103° F. Respiration was 24 on admission, but the next day was 32. The wound was not infected. There was some lagging in breathing on the right side; the chest below the wound was tympanitic, with decrease in breath sounds; tactile fremitus was unchanged. Cellular emphysema was absent. The morning after admission, dulness was present in the right chest over the lower ribs in the postaxillary line, and it was thought that a penetrating wound existed, possibly with hemopneumathorax. The roentgenologic report the following day revealed no evidence of pneumathorax or hemothorax. The patient later developed a cough, but had no bloody expectoration. His condition improved constantly, the temperature reaching normal on the fifth day. Treatment consisted in bed rest, care of the wound, cough medicine and two hypodermics of morphia. Dismissal on the tenth day, apparently well. The final diagnosis was penetrating wound of the chest, with hemopneumathorax too small to be detected by roentgenology.

Causes of Penetrating Wounds.—Of the 1,009 cases of penetrating wounds, 799 (79 per cent) were stab wounds, and 207 (21 per cent) were

gunshot wounds. Two patients were injured in automobile accidents, and one by a falling roof. This class of patients is not so subjected to automobile accidents as white patients. Injuries from automobile accidents are more likely to be nonpenetrating than penetrating, but it is well to recognize the fact that the same kind and same extent of injuries may take place in wounds which do not penetrate the thoracic cavity as in those which produce penetration. Fatal damage may occur to the lungs or pleura by concussion or compression without a penetrating wound. However, the possible presence of open pneumathorax in a penetrating wound increases the gravity of the prognosis in such cases.

Signs and Symptoms.—In the average patient with penetration, the injury was accompanied by more or less pain, followed by weakness, and in severe cases by shock. The majority of the patients reached the hospital within an hour or two after being hurt, although a delay of three days is recorded in one case. Insistent persuasion sometimes is necessary to induce sick Negroes to enter the hospital, that is, those with appendicitis or intestinal obstruction, or afflicted with various medical disorders; but those with trauma, such as fractures and gunshot wounds, usually come voluntarily. Another characteristic of Negro patients is noticed in their apparent indifference to traumatic lesions as compared with their apprehension in mysterious internal maladies. Such lack of psychic reaction probably is an important factor in the favorable results obtained in chest injuries.

Cough and hemoptysis in the average patient were signs of uncertain value. Their absence was not regarded as significant. Sometimes there was a history of expectorating a considerable amount of blood immediately after the injury, which did not appear again. Hemoptysis does not necessarily indicate laceration of lung tissue, nor is it a constant sign of the lesion. Hemoptysis rarely is fatal unless one of the large blood vessels of the hilus is ruptured, and there is direct communication between a bronchus and the vessel, or unless there is extensive laceration of a lung which is unable to collapse on account of adhesions.

Dyspnea generally was present to a greater or less extent. Marked distress in breathing usually meant pneumathorax or hemothorax. In the early stages of the typical case two characteristic signs of penetrating wounds are lagging of the affected side on respiration, and the presence of moist râles in the area involved. As a rule increased pulse and respiratory rate, fever and leukocytosis were present from the beginning, although mild cases might barely show such signs. Decreased resonance and diminished breath sounds were noted until the presence of air caused increased resonance or the presence of fluid caused dulness. Cyanosis is difficult to recognize in these patients.

Pneumathorax, Hemothorax, Hemopneumathorax.—Pneumathorax was diagnosed in 193 cases (19 per cent); hemothorax in 248 cases (25 per cent); hemopneumathorax in 382 (38 per cent); and none of the above in the remaining 18 per cent. It is probable that hemopneumathorax was present

in even a larger percentage of cases than is indicated. Sometimes the amount of air was enough to cause complete pulmonary collapse, while in one case as much as 2,700 cc. of bloody fluid was aspirated in one sitting. The largest total amount of fluid removed in one patient was 10,900 cc., taken in quantities from 500 to 1,400 cc. over a period of five weeks. The extent of air or blood present may be limited by preexisting pleural adhesions.

The signs and symptoms of hemopneumathorax resemble those of hydrothorax, with certain differences. In the former dyspnea almost always is present, and is intense. The accumulation and absorption of the blood in some cases were responsible for a rise of temperature as high as 103° F. This evidently was a so called aseptic fever as proved by its subsidence upon withdrawing the blood. Occasionally the dulness and resistance in hemopneumathorax were not so pronounced as one would expect with the displacement of the heart and the dyspnea. Such lack of dulness and resistance was supposed to depend upon the mixing of air with the blood. Air was aspirated in nine of the cases in the series, and blood was aspirated in 185 cases (18 per cent).

Roentgenologic Evidence.—Often the first roentgenologic evidence of penetration was slight elevation of the diaphragm on the affected side. It is interesting that this sign should exist even with a large hemothorax, the weight of which should lower the level of the diaphragm. Two explanations for the phenomenon have been offered,[2] one being that the phrenic nerve, which is comparatively unsupported in its passage through the thorax, is particularly susceptible to the concussion produced by the injury, and that paralysis of the nerve permits the elevation. The other suggestive explanation is that the collapse of the lung following the impact of the foreign body causes at first a considerable negative intrapleural pressure. The fluoroscopic view and roentgenogram are of indispensable aid in studying thoracic injuries. They are always correct, but sometimes we read in them things which are not there, or do not read things which are present.

Infection.—Various authors and text-books continue to list infection as a common complication of penetrating wounds of the lungs and pleura. Possibly war wounds are meant. Infection was extremely rare in this series. There were only 17 cases of empyema, with six deaths, and no instances of pulmonary abscess or gangrene. Eight cases of pneumonia were reported, with five deaths. Some wounds of the chest wall became infected, but seldom with serious results. Infection in such wounds and in wounds in other parts of the body were far commoner than infection in the pleura. The pleura seems to be endowed with as effective resistance against ordinary infection as the peritoneum.

Cellular Emphysema and Sucking Wounds.—Cellular emphysema was noted in 159 cases (15 per cent). Sometimes there was only a small amount of air in the tissues about the chest wound; again most of the chest wall, axilla and neck were involved. Emphysema does not necessarily indicate penetration, and the same may be said of the so called sucking wound. Air

from the outside may enter the tissues through a wound in any part of the body, but particularly the thick muscular tissues of the chest wall, on account of the suction action of the movements of respiration. Likewise a nonpenetrating wound of the chest wall may furnish the appearance and sound of a sucking wound due to communication with the pleural cavity.

Treatment.—The treatment of the majority of patients was simple, consisting of sterilization of the wound, or wounds, with débridement if indicated, bed rest, and the administration of ample sedatives. Usually the chest was strapped, but if the patient objected, strapping was omitted, even if ribs were fractured—a rare complication. Sucking wounds were sutured immediately. The majority of the patients received tetanus and gas bacillus antitoxin. No case of tetanus or gas bacillus infection developed.

The administration of saline solution and glucose subcutaneously or intravenously as routine treatment in all but the mildest cases has proved of material assistance in overcoming shock and hastening convalescence. Formerly it was difficult to obtain donors for blood transfusion in Negroes, but since they have learned that no harm comes to the donor, the transfusion is performed far more frequently, and is especially valuable in patients with recurring hemothorax.

The most serious consequence of thoracic trauma is hemorrhage. Fortunately the blood pressure in the pulmonary system is lower than that in the general circulation, and pulmonary blood is said to clot more readily; otherwise death from hemorrhage would result in a larger number of the cases. Believing that the presence of air and blood in the pleural cavity acts as an effective tamponade against further bleeding, it has become a rule in the Grady Hospital not to aspirate blood during the first 48 hours after the injury, unless the patient complains of distressing pain or dyspnea.

Rupture of the blood vessels of the thoracic wall calls for prompt ligature. Such hemorrhage occurring externally is easily recognized, but bleeding into the pleural cavity may be mistaken for pulmonary hemorrhage. In this series there were five ligations of an intercostal artery, with two recoveries, and two ligations of the internal mammary artery, without recovery. Exploratory thoracotomy for pulmonary hemorrhage was done once, with failure to save the patient. Some form of interruption of the phrenic nerve to elevate the diaphragm has been suggested by Warner[3] as an aid in controlling intrathoracic bleeding.

So few operative procedures were employed in this large group of chest injuries that the series almost falls into the category of medical rather than surgical cases. The results, however, appear to justify such conservative treatment. It is admitted that a few of the fatalities in this record might have been avoided by early thoracotomy. The danger in protracted satisfactory results in the nonoperative treatment is that when a case is presented which calls for radical management, the different situation is not realized, or realization comes too late.

Blood was aspirated, under the rules mentioned, in 185 cases (18 per

cent); air was aspirated in nine cases. The diaphragm was sutured in 18 cases, with ten recoveries. It was not feasible to suture the diaphragm in one instance, on account of the patient's weak condition. He finally was discharged as improved, with a potential diaphragmatic hernia.

Mortality.—The total number of deaths among the 1,009 cases was 136 (13 per cent), gunshot wounds proving more fatal. Deducting the patients who died of irremediable trauma within the first 24 hours after admission, 46, the deaths numbered 90, a mortality rate of 9 per cent. Fifty patients came in with associated injuries of the abdomen, kidneys, spinal cord and other viscera, which contributed to the fatal result. Deducting this number leaves 86 patients whose demise was attributed solely to chest injury, a mortality of 8.5 per cent.

End-results.—In this class of patients a follow up system is practically impossible. When they leave the hospital they are requested to return to the outpatient clinic for observation, but very few ever return. It is difficult to locate them at home since they constantly move and change their names. Many of the patients owe their injury to law breaking, and they do not care to coöperate in a follow up system. They fear the police are still on their trail. In spite of so many patients being discharged as well or improved, no doubt an investigation of the end-results would reveal crippling permanent disabilities from pleural adhesions and other factors.

Summaries of two typical bad cases are submitted, with roentgenographic illustrations:

CASE REPORTS

Case 2.—Male, aged 27, admitted to the hospital August 5, 1935. Two or three hours previously he had received a stab wound in the upper left chest anteriorly, accompanied by considerable pain and a large amount of bleeding. When first seen in the ward he was suffering from respiratory distress and mild shock. Pulse on admission was 90, temperature 97.3° and respiration 24. Emphysema was present at the base of the neck and in the upper part of the chest. There were lagging respiration on the affected side, and diminished breath sounds. The next day the pulse was 120, temperature 101°, the respiratory note remaining about the same throughout sojourn in the hospital. The pulse did not go any higher, and the temperature ranged between 99° and 103° and 104° during four months' hospitalization. The leukocyte count was normal except in the beginning of empyema, when it rose to 14,000. Dulness soon developed in the left side, and the roentgenogram three days following admission indicated fluid forming. Four weeks later aspiration withdrew 100 cc. of serosanguinous fluid. The patient was given 500 cc. of blood by transfusion, and a week later rib resection was performed, and 800 cc. of thick pus were removed. He was dismissed as improved.

Case 3.—An example of a patient admitted to the outpatient clinic for suture of an apparently mild injury of the chest wall, allowed to go home, and having to enter the hospital ten days later on account of weakness, bleeding and shortness of breath. He was 31 years old, and when first seen had to sit up in bed on account of dyspnea. Expansion was limited on the affected side, the percussion note was dull, and there was absence of tactile fremitus. He was admitted May 8, 1935, and two days later the roentgenogram disclosed hemothorax of the left chest with complete displacement of the mediastinal contents to the right. The same day 1,000 cc. of bloody fluid were

aspirated, and specimen sent to laboratory for culture, which at this time was negative. However, the patient's temperature ran a septic course until death, after 48 days. The total quantity of fluid aspirated in five weeks was 10,900 cc., in amounts from 500 to

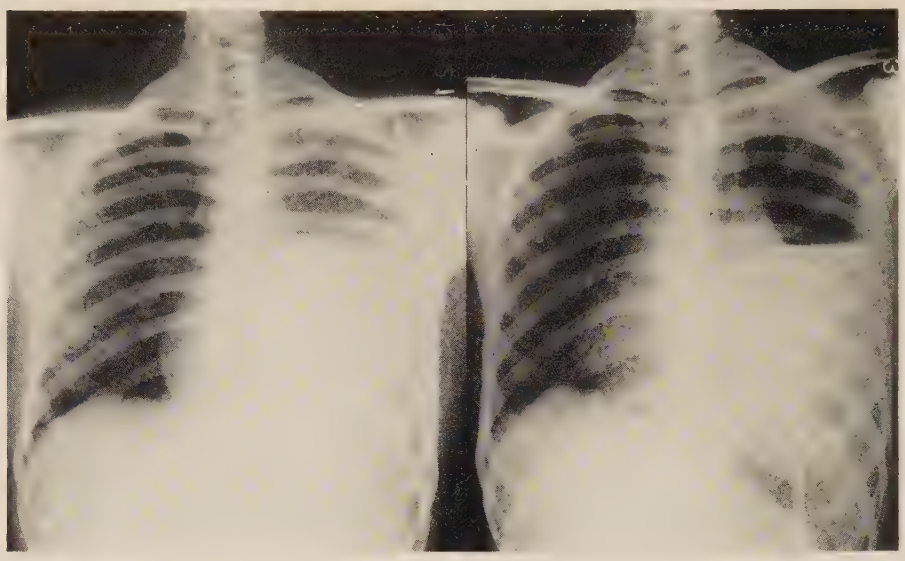

FIG. 1.—Case 2.—First Film. Showing uniform increased density over lower two-thirds left chest, with fluid level rising in the axilla and over the apex. No fluid level to suggest pneumathorax. Heart not displaced. Impression: hydro- or hemothorax left chest.

FIG. 2.—Case 2.—Second Film. Showing pyopneumathorax left chest, with fluid level opposite seventh intercostal space in scapula line. Left lung collapsed, heart displaced to right. Drainage tube in fluid space.

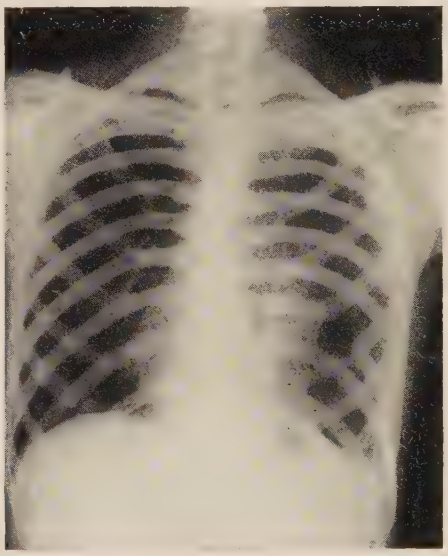

FIG. 3.—Case 2.—Third Film. Showing pneumathorax left chest, with about 60 per cent lung collapsed. Ninth left rib has been resected in posterior axillary line. Parietal pleura thickened, heart still displaced to right. Small amount of fluid still present. Impression: postoperative empyema. Patient recovered.

1,400 cc. The character of the fluid gradually changed from blood to pus. The patient was given one blood transfusion three days after entering the hospital. Rib resection for empyema was performed five weeks later. An early thoracotomy for checking hemorrhage might have offered the patient a better chance.

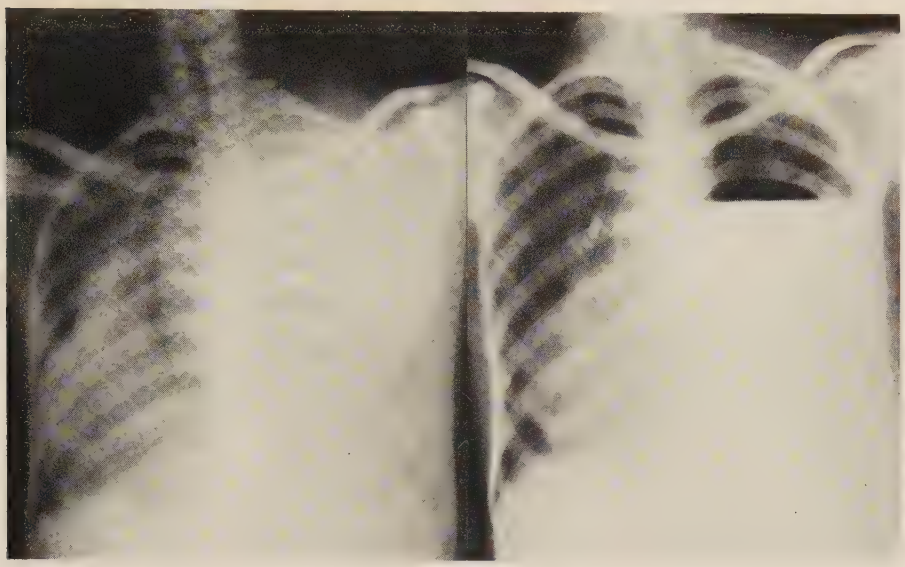

FIG. 4.—Case 3.—First Film. Showing hemothorax, with complete displacement of heart to right. Note displacement of trachea.

FIG. 5.—Case 3.—Second Film. Showing hemothorax, with fluid level extending to third interspace anteriorly. Pneumathorax above fluid level. Heart displaced to right. Total of 10,900 cc. of fluid removed at different aspirations. Patient died.

SUMMARY AND CONCLUSIONS

Penetrating wounds of the lungs and pleura may exist with so little pneumathorax or hemothorax as not to be demonstrable in the roentgenogram. Empyema and other serious infections result in less than 2 per cent of the cases. Hemorrhage is the commonest cause of death. The chest wall should be examined for bleeding. Pain and dyspnea are relieved by withdrawing blood and air, but intrathoracic hemorrhage may be controlled by deferring aspiration for 48 hours. The nonoperative treatment of the ordinary cases encountered in civil practice gives a low mortality rate, but in a few cases radical surgery is urgently indicated. The total mortality rate in 1,009 cases was 13 per cent; deducting the patients who died in the first 24 hours, the rate was 9 per cent; deducting the patients who had associated injuries which contributed to their deaths, the rate was 8.5 per cent. The final effects of these injuries should be studied.

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A TEN YEAR STUDY OF EMPYEMA IN CHILDREN

1926–1936

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EMPYEMA as a surgical disease has been of great interest to the surgeon for many years. The greatest progress in the management and refinement of technic has been evolved since the initial work of the Empyema Commission[1] which was established by the Surgeon General of the Army in 1918. The basic principles as emphasized by this Commission still form the foundation of our present day therapy.

In the hope of lowering the mortality of this disease many and varied details of treatment have been advocated by numerous authors. The principle of avoiding the creation of an open pneumathorax in the formative stage of an empyema is fundamental and is now a universally accepted fact. There is a present day trend in advocating open drainage over all other methods when the empyema is a true abscess. This procedure is founded on the experience of many surgeons[2, 3, 6] as meeting the most satisfactory requirements of producing good results.

The studies herein reported include a method of surgical drainage which combines the principles of closed and open drainage. They also include a classification of the type of pneumonia preceding the empyema and the incidence of the latter. Being impressed by the studies of Heuer,[4] Graham and Berck[2] in which they noted a parallelism of the mortality of pneumonia and empyema, we were led to review our records in this light.

In the ten year period 1926 to 1936, 5,868 patients were classified as pneumonia, and of this number 407 or 0.7 per cent developed empyema as a complication which required surgical drainage. In this series a similar parallelism has been found as previously reported by the above authors and lends further support to the idea expressed that the type or virulence of the pneumonia preceding the empyema is the most important factor in determining the mortality rate of empyema in a given series treated over a period of years. It is interesting to note in this ten year period that empyema occurred as a complication in 8.9 per cent of the patients classified as lobar pneumonia and in only 1.2 per cent of those classified as bronchopneumonia (Chart 1).

The mortality of lobar pneumonia for this period was 14.3 per cent, while that of bronchopneumonia was 44.5 per cent (Chart 2).

Another point of interest is that the highest mortality of empyema in St. Louis is reported by Graham in 1926, while that reported by Heuer from Cincinnati was highest in 1927, and in Detroit our highest mortality was in 1929 (Chart 3).

This observation shows that the same disease has a different mortality in spite of the fact that the treatment was a uniform procedure in these different cities. This may be explained by the variance of virulence of the epidemic of the preceding pneumonia in relation to geographic location.

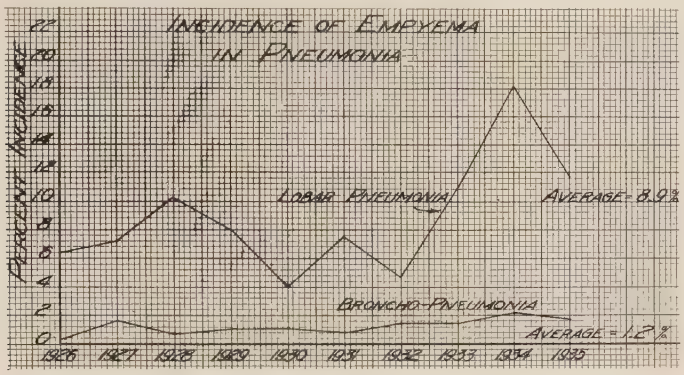

CHART 1.—Incidence of empyema in pneumonia by year periods.

A uniform procedure of treatment has been practiced in the surgical drainage of these cases consisting of aspiration up to the stage when frank pus is obtained. At this time the trocar-cannula-catheter method of closed drainage is instituted under local anesthesia. Closed drainage is maintained by apply-

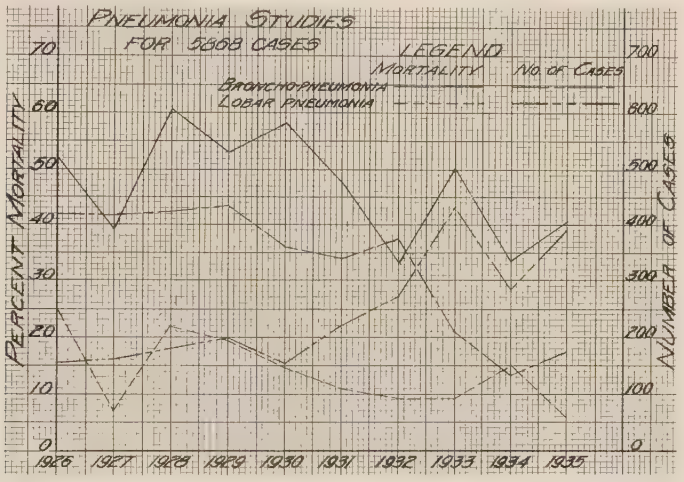

CHART 2.—Number and mortality rate of pneumonia by year periods.

ing a hemostat to the end of the catheter. The empyema cavity is aspirated in the operating room up to the point where reflex coughing or a small amount of blood-tinged pus appears in the fluid. The patient is then returned to the ward. There has not been any evidence of shock or circulatory or respiratory distress. Further aspirations are carried out every four hours by the house officers or graduate nurses trained in this technic. The majority of cases in the early years of this series were subsequently irrigated with 0.5 per cent

sodium hypochlorite solution (Dakin's solution) following aspiration of the pus, but of late years, irrigations have been used less often with no material difference in the prolongation of the period of morbidity. It is observed, as reported by others,[3] that with this type of closed drainage there may be some

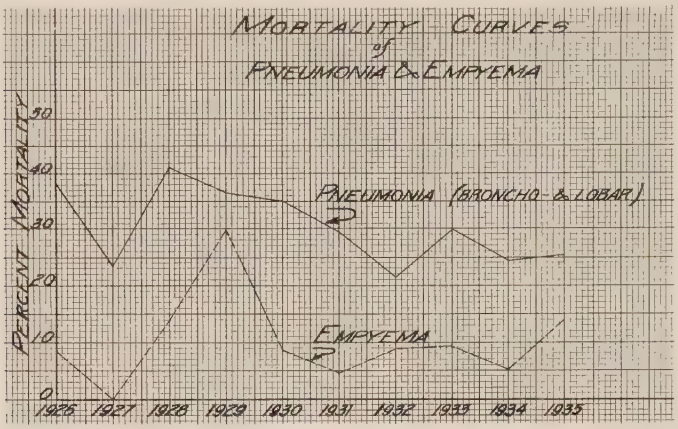

Chart 3.—Mortality curves of pneumonia and empyema by year periods.

leakage of pus around the tube, but this occurs at a time when the empyema cavity is practically empty and should be no cause for alarm or for the changing of the catheter. The original catheter is left in place usually 12 to 18 days, at which time it is cut and allowed to remain open as a drain. In the

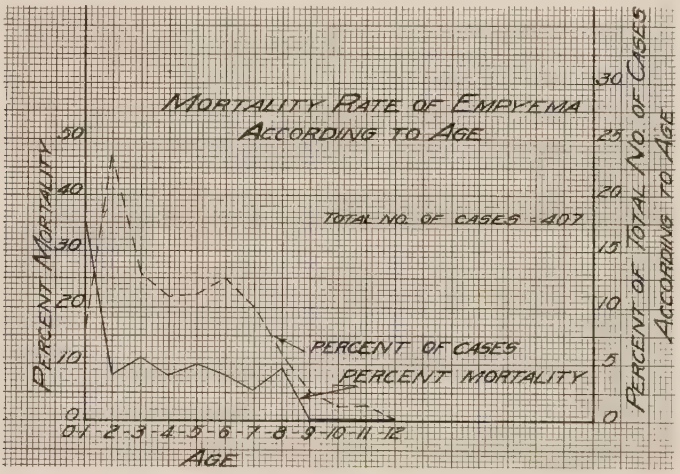

Chart 4.—Mortality rate of empyema according to age of patients.

event there is clinical evidence of retention, manifested by a rise in temperature or roentgenologic evidence of fluid, the catheter is replaced by a larger tube. It may be necessary to use local or nitrous oxide-oxygen anesthesia before inserting this larger tube. This lessens the mental trauma to a child who has been seriously ill. It has not been necessary in our experience to

resort to rib resection for adequate drainage following the procedures outlined except in the following instances:

(1) Four patients developed recurrent empyema after the procedure described above who later made good recoveries.

(2) Eleven other patients required rib resection with open drainage:

(*a*) In five patients the pus was too thick for a catheter drainage as initial treatment.

(*b*) Five infants (1926–1927)—reason not recorded.

(*c*) One—reason not recorded.

In this group of 15 patients the average mortality was 6.7 per cent (one death) and a mean period of morbidity of 78.9 days.

Factors Influencing Mortality

(1) As previously emphasized, the mortality of empyema bears a definite relationship to the type or virulence of pneumonia preceding the empyema.

(2) The age of the patient, as statistics show, that the greatest mortality is in infants, two years or younger. This is shown also in this series. The mortality for the various age groups up to 12 years is shown in Chart 4, as is also the percentage of cases according to the age groups.

(3) Poor nutrition of the patient is an important factor in any given series.

Factors Influencing Morbidity

The average hospital stay was 48.6 days (Chart 5).

This period of morbidity may appear unusually long where early treatment is instituted and carefully followed. It can be explained by certain definite factors, *viz:*

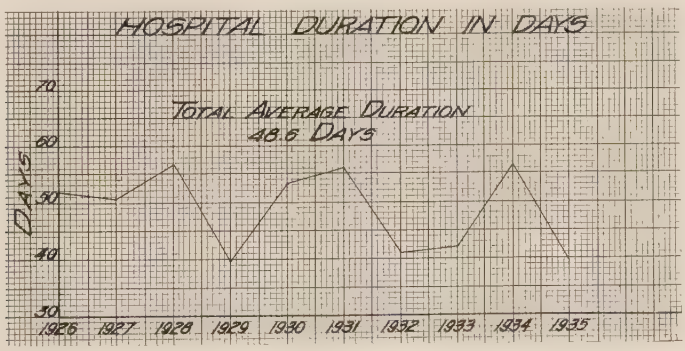

Chart 5.—Average hospital stay of patients by year periods.

(1) Many of these patients were admitted late in the stage of their illness.

(2) The nutritional status of most of these patients entering this hospital is below normal and their apparent reaction to their infection is impaired.

(3) The virulence of the organism.

(4) Multiple encapsulations.

(5) Complications such as otitis media, mastoiditis, bronchial fistula, *etc.*

(6) It has been the practice to observe the patient in the hospital for at least five days after the removal of the tube. Experience has shown that the patient who is sent home with a discharging sinus is frequently readmitted to the hospital because of a reaccumulation of fluid. An additional five day period in the majority of cases is established as an added precaution before discharge because of the poor home conditions of many. No patient has been discharged until the temperature remains normal, the roentgenologic check-up shows no fluid, or pneumathorax, and the sinus is free from drainage.

(7) In those patients with a large pneumathorax persisting after removal of the pus and in whom reexpansion of the lung has been slow, we have materially shortened the period of morbidity in the past two years by applying the Wangensteen[5] method of suction.

End-Results

Four hundred seven patients were treated by a combined form of closed and open method of surgical drainage with an average mortality of 10.3 per cent (Chart 6).

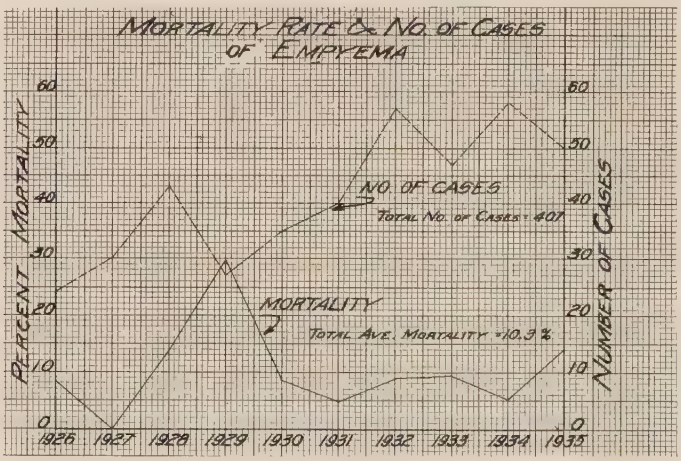

Chart 6.—The mortality curves of pneumonia and empyema by year periods.

Of the 365 patients who survived, all were clinically cured of the empyema except three. The latter required major operative intervention. One was classified as tuberculous empyema with a mixed infection, in whom closed drainage, followed by thoracoplasty in two stages, gave a good clinical end-result. One patient required an unroofing with a subsequent good result, while the remaining patient necessitated a decortication. The latter patient died 12 hours postoperative of surgical shock. Fifteen patients required rib resection and open drainage as previously described.

SUMMARY

(1) A ten year review of pneumonia and empyema cases is reported.

(2) There is a definite parallelism of the mortality of pneumonia and empyema.

(3) A uniform procedure of surgical drainage combining the closed and open technic is presented.

(4) Four hundred seven cases of empyema were treated by this procedure with an average mortality of 10.3 per cent.

(5) Of the 365 patients who survived, all but three made excellent clinical recoveries on discharge from the hospital.

(6) Fifteen patients required rib resection and open drainage.

(7) The average hospital stay of these patients was 48.6 per cent days.

(8) The Wangensteen method of suction offers a valuable aid in shortening the period of morbidity due to failure of the lung to reexpand after the surgical drainage of an empyema.

(9) Three patients required major surgical procedures for chronic empyema.

CONCLUSIONS

(1) The combined interest of the pediatrician, roentgenologist, and surgeon is important in the careful management of a child ill with empyema.

(2) Careful clinical and roentgenologic examination (anteroposterior and lateral positions).

(3) Aspiration for diagnostic (pus to be cultured) and therapeutic purposes up to the point of frank pus.

(4) The combined method of trocar-cannula-catheter drainage followed by open drainage is recommended.

(5) Attention to details which include blood transfusions as indicated, preservation of the normal water balance and nutritional status of the patient are essential to the successful management of a case of empyema.

We wish to express our thanks to the members of the medical department for their kind cooperation in this work and especially to Dr. Hira E. Branch for his valuable assistance in preparing the statistical data for this review.

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GASTRIC ACIDITY FOLLOWING OPERATIONS FOR GASTRIC AND DUODENAL ULCER

ITS EFFECT ON THE QUESTION OF PARTIAL GASTRECTOMY

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DIVISION OF SURGERY, THE MAYO CLINIC

"GASTRIC ulcer and biliary disorders likewise present distinct regional differences—the part played by racial factors in these differences is as yet unknown to science." This statement recently appeared in a review of Schittenhelm's[1] work on the distribution of disease in Germany.

In 1930, after Snell and I had visited various surgical clinics abroad, particularly those in central Europe, we published a series of papers[2, 3, 4, 5] describing and illustrating the difference between the lesions of duodenal ulcer for which we saw operations performed in Germany and those for which it has been my privilege to operate at The Mayo Clinic. This difference lay largely in the high degree of gastritis associated with duodenal ulcer of German patients operated upon at German surgical clinics, as compared with the low incidence of associated gastritis in a series of patients operated upon at The Mayo Clinic. Accuracy of these observations was confirmed by Sebening,[6, 7] of Schmieden's Clinic (Frankfort am Main), who spent several months in study at The Mayo Clinic. More recently, in a clinical study, Church and I[8, 9] showed that gastritis was a frequent accompaniment of pyloric obstruction. Dragstedt's[10] experimental work confirmed our observations. Obviously, if gastritis is lacking to any great degree in a percentage of cases in which operation is performed for duodenal ulcer, gastritis cannot be considered an indication for subtotal or partial gastrectomy as a routine procedure in the treatment of duodenal ulcer. When I was discussing with von Haberer,[11] of Cologne, this difference of lesions accompanying duodenal ulcer, he made the interesting statement that in his experience as surgeon in Vienna, Graz, Dusseldorf, and Cologne, he found marked differences in the types of lesions encountered in these different areas. This was true even when such short distances separated the cities as that which lies between Dusseldorf and Cologne. These factors of so called geomedical variations may account, in part, for the difference in the results experienced in the treatment of different diseases in various countries, indeed, in different areas of the same country.

Concerning the surgical treatment of duodenal ulcer, it has been stated that ulceration does not recur after relative achlorhydria has been established and that failure to obtain relative achlorhydria following gastric resection for duodenal ulcer is attributable to the fact that an insufficient amount of

stomach has been removed. Although this was heard more frequently ten years ago than now, more recent experience of one group of investigators in the United States would indicate that relative achlorhydria occurred in but approximately 50 per cent of a series of 108 cases in which operation was performed in the clinic[12] where these investigators work. In this same reported series, nine cases of gastrojejunal ulcer developed in a group of 47 cases in which persistent relative achlorhydria was not established subsequent to subtotal gastrectomy.

In order to add further to knowledge of the effect of various operations for gastric and duodenal ulcer, studies of gastric acidity, using Töpfer's method, have been carried out in a group of approximately 150 cases encountered at The Mayo Clinic. In these cases, in the past year and one-half,

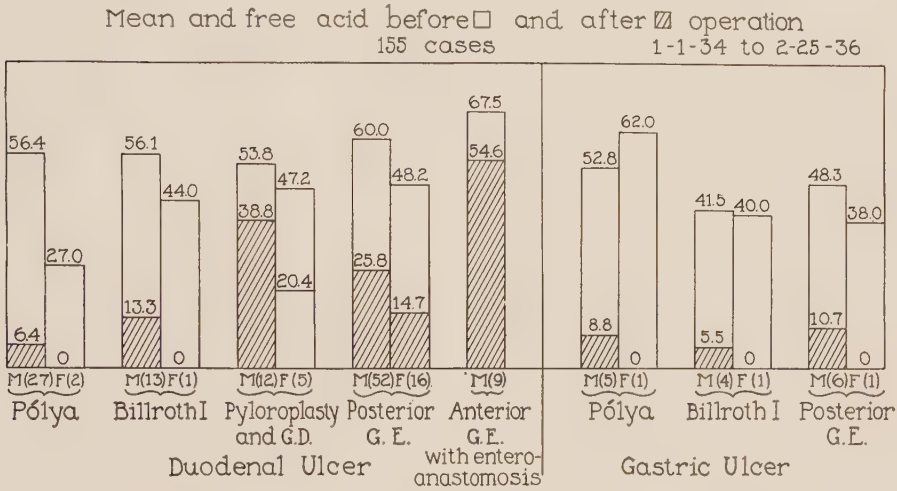

Chart 1.—Reduction of gastric acidity following various operations for gastric or duodenal ulcer.

I performed gastro-enterostomy, pyloroplasty, or gastric resection of the Billroth I or Pólya type for one or the other of the conditions named. Reduction in gastric acidity usually occurs following all of these procedures although most frequently, and to the greatest extent, following gastric resection of the posterior Pólya[13] type (Chart 1). When anterior gastro-enterostomy is performed for duodenal ulcer, combined with entero-anastomosis, the latter preventing regurgitation of any great amount of duodenal and jejunal secretion into the stomach, very little reduction of gastric acidity takes place (Chart 1 and Table I). In an occasional case, when entero-anastomosis has been part of a gastric resection of the Balfour-Pólya type, similar lack of reduction in gastric acidity may occur. In many cases in which posterior gastro-enterostomy is performed for duodenal ulcer, gastric acidity will be reduced to the point of relative achlorhydria (Tables II and III). It would seem, therefore, that reduction of gastric acidity subsequent to posterior gastro-enterostomy and gastric resection of the Pólya type, in

TABLE I

PARTIAL LOSS OF THE DILUTION OF GASTRIC ACIDITY WHEN ANTERIOR GASTRO-ENTEROSTOMY WITH ENTERO-ANASTOMOSIS IS PERFORMED FOR DUODENAL ULCER

Case	Age	Sex	Duration of Symptoms	Character of Ulcer	Acidity			
					Preoperative		Postoperative	
					Total	Free HCl	Total	Free HCl
1....	33	M.	15 Months	Subacute perforating	50	36	28	20
2....	52	M.	6 Months	Subacute perforating	98	90	82	70
3....	31	M.	12 Years	Subacute hemorrhagic perforating	62	46	60	44
4....	59	M.	3 Years	Subacute perforating	90	80	94	84
5....	65	M.	25 Years	Subacute perforating	58	46	74	56
6....	35	M.	15 Years	Hemorrhagic perforating	88	78	84	70
7....	56	M.	12 Years	Subacute perforating	92	84	60	50
8....	38	M.	25 Years	Multiple subacute perforating	60	50	50	38

many cases is the result of dilution of the gastric secretion by the fluids regurgitated from the attached jejunal loop. Further evidence of this is the fact that, in many cases in which achlorhydria seems to have occurred subsequent either to gastric resection or gastro-enterostomy for duodenal ulcer, acid gastric secretion can be obtained after injection of histamine.

TABLE II

RELATIVE ACHLORHYDRIA OCCURRING IN SOME CASES OF DUODENAL ULCER AFTER GASTRO-ENTEROSTOMY

Case	Age	Sex	Duration of Symptoms	Character of Ulcer	Preoperative Acidity		Histamine Interval, Minutes							Postoperative Acidity	
					Total	Free HCl	10	20	30	40	50	60	70	Total	Free HCl
1....	24	F.	3 Years	Subacute perforating with obstruction	40	34								14	0
2....	38	M.	20 Years	Subacute perforating	62	50								12	0
3....	48	M.	12 Years	Subacute perforating obstructing	76	70	Total							8	0
							26	48	70	80	92	76	100		
							Free HCl								
							16	42	62	74	86	70	96		
4....	35	M.	20 Years	Acute perforating	100	80								14	0
5....	47	M.	5 to 6 Years	Subacute perforating	118	100								10	0
6....	47	M.	35 Years	Chronic (?)	60	40								16	0

There are some cases, particularly after the Pólya type of resection, in which the histamine will not produce any response in gastric acidity. Further support for the belief that dilution plays an important part in reduction of gastric acidity following operative procedures for duodenal ulcer is the fact that when pyloroplasty has been performed, reflux of duodenal secretion is not active because of the onward normal peristaltic movement of fluids from the stomach through the duodenum and in such cases reduction in gastric acidity is obtained less frequently than when gastric resection or

TABLE III

RELATIVE ACHLORHYDRIA OCCURRING IN SOME CASES AFTER EXCISION OF GASTRIC ULCER AND GASTRO-ENTEROSTOMY

Case	Age	Sex	Duration of Symptoms	Character of Ulcer	Acidity			
					Preoperative		Postoperative	
					Total	Free HCl	Total	Free HCl
1.....	46	M.	6 Years	Subacute	52	40	12, 12, 28, 28 0, 0, 0, 0 0, 0, 20, 20	
2.....	45	M.	18 Months	Chronic pyloric	80	70	16	8
3.....	43	M.	15 Years	Chronic (?)	72	60	8	0
4.....	48	F.	12 Years	Chronic (?)	50	38	8	0
5.....	38	F.	5 Years	Chronic	Not estimated	Not estimated	8	0
6.....	59	F.	1½ Months	Subacute perforating gastric and chronic duodenal	Not estimated	Not estimated	14	4
7.....	64	M.	20 Years	Subacute perforating	74	60	10, 12, 20	0, 0, 0
8.....	35	M.	7 Years	Subacute	36	26	4	0
9.....	64	M.	20 Years	Subacute perforating	74	60	10	0
10*....	42	M.	3 Years	Perforating	54	42	48	36

* Gastrojejunal ulcer developed.

gastro-enterostomy are performed. Similarly, even in cases in which extensive gastric resection has been performed for duodenal ulcer, and the end of the stomach has been sutured directly to the duodenum, the so called Billroth I von Haberer type of anastomosis (Table IV), the percentage of patients who will obtain relative achlorhydria is not great in proportion to those patients with duodenal ulcer who have been subjected to a Pólya type of anastomosis subsequent to gastric resection.

Gastric ulcer, on the other hand, apparently differs from duodenal ulcer, not only pathologically, but in its biologic response to the operative procedures mentioned in the preceding paragraph. Following gastric resection

TABLE IV

THE INFREQUENT OCCURRENCE OF RELATIVE ACHLORHYDRIA WHEN BILLROTH I-HABERER ANASTOMOSIS FOLLOWING PARTIAL GASTRECTOMY AND PARTIAL DUODENECTOMY IS PERFORMED FOR DUODENAL ULCER

Case	Age	Sex	Duration of Symptoms	Character of Ulcer	Acidity Preoperative Total	Acidity Preoperative Free HCl	Acidity Postoperative Total	Acidity Postoperative Free HCl
1.....	49	M.	15 Years	Subacute perforating hemorrhagic duodenal	46	32	18	8
2.....	56	M.	8 Years	Duodenitis	62	52	8	0
3.....	33	M.	17 Years	Gastrojejunal ulcer healed. Duodenal	50	38	16	0
4.....	47	M.	15 Years	Perforating duodenal	70	64	54	40
5.....	32	M.	10 Years	Multiple duodenal with duodenitis	96	80	40	36
6.....	59	M.	30 Years	Chronic bleeding duodenal	40	20	32	20
7.....	35	M.	14 Years	Multiple subacute duodenal	34	24	56	44
8.....	27	M.	5 Years	Multiple chronic duodenal with duodenitis	88	78	28	20

of either the Pólya or the Billroth I type for gastric ulcer, reduction of gastric acidity to a degree of relative achlorhydria occurs in practically every case (Tables V and VI), whereas similar surgical procedures, in which equally large amounts of stomach are removed for treatment of duodenal ulcer, are

TABLE V

RELATIVE ACHLORHYDRIA FOLLOWING PARTIAL GASTRECTOMY OF BILLROTH I TYPE

Case	Age	Sex	Duration of Symptoms	Character of Ulcer	Acidity Preoperative Total	Acidity Preoperative Free HCl	Acidity Postoperative Total	Acidity Postoperative Free HCl
1.....	53	M.	7 Months	Gastric	56	44	6	0
2.....	55	M.	9 Months	Chronic hemorrhagic perforating gastric	70	58	10	0
3.....	57	M.	4 Months	Ulcerating gastric lesion	64	52	8	0
4.....	49	M.	3 Months	Chronic gastric	52	40	60	0
5.....	57	M.	6 Weeks	Subacute perforating gastric and duodenal	54	30	8	0
6.....	58	M.	4 Years	Chronic gastric	28	16	36	22
7.....	50	M.	30 Years	Subacute perforating gastric	6	0	12	0
8.....	55	F.	15 Years	Perforating gastric	52	40	10	6

followed in only approximately 50 per cent of cases by relative achlorhydria.[11] Interesting in this connection is the fact that after excision of a gastric ulcer and gastro-enterostomy relative achlorhydria will develop in a much higher

percentage of cases than when gastro-enterostomy has been performed for duodenal ulcer. This probably accounts for the fact that gastrojejunal ulcer very seldom occurs following operative procedures for gastric ulcer, providing increased rapidity of emptying of the stomach and dilution of gastric secretion by jejunal secretion are obtained.

TABLE VI

RELATIVE ACHLORHYDRIA FOLLOWING PARTIAL GASTRECTOMY OF POSTERIOR PÓLYA* TYPE FOR GASTRIC ULCER

Case	Age	Sex	Duration of Symptoms	Character of Ulcer	Acidity Preoperative Total	Acidity Preoperative Free HCl	Acidity Postoperative Total	Acidity Postoperative Free HCl
1.....	47	M.	2½ Years	Subacute perforating gastric	76	60	4	0
2.....	58	M.	4 Years	Multiple gastric	48	30	20	0
3.....	44	M.	8 Years	Recurring subacute duodenal and gastric?	46	34	0	0
4.....	58	M.	Several Months	Subacute perforating gastric	60	42	10	0
5.....	56	M.	1½ Months	Subacute gastric perforating ulcerating			8	0
6.....	57	M.	25 Years	Obstructing gastric	40	30	5	0
7.....	40	F.	31 Years	Chronic gastric; chronic duodenal	38	24	4	0
8.....	58	M.	3 to 4 Years	Chronic perforating gastric	60	52	5	0

* Hoffmeister-Pólya type in Case 5.

Studies of preoperative and postoperative gastric acidity, it seems to me, have an important bearing on surgical treatment, both of gastric and of duodenal ulcer, but more particularly of the latter. Since gastrojejunal ulcer does not develop, except in rare cases, in the presence of achlorhydria, those methods which produce anacidity would appear to be superior to others, providing the risk of the procedure was not too great. Unfortunately, the risk of gastric resection, combined with resection of enough of the duodenum to remove the duodenal ulcer, is several times greater than that of gastro-enterostomy or pyloroplasty.[14] This is largely attributable to the difficulty and risk of removal of large or multiple extensive duodenal ulcers as a part of the operation of partial gastrectomy. Other things being equal, the risk in such cases is directly proportional to the size and extent of the ulcer and to the difficulty of closure of the end of the duodenum after removal of part of it. If, therefore, partial gastrectomy for duodenal ulcer can be counted upon to produce relative achlorhydria in only 50 per cent of cases, at a risk considerably greater than that of gastro-enterostomy, and if gastro-enterostomy will reduce gastric acidity to a varying degree in all cases, even to the point of relative achlorhydria in some cases, it would appear that the

additional risk of partial gastrectomy as a routine procedure in the treatment of duodenal ulcer requires justification on some basis other than that of the production of relative anacidity. Such a statement, however, must not be construed as detracting from the advantage of partial gastrectomy in selected cases of duodenal ulcer, particularly those in which the ulcer is of hemorrhagic type, nor in that group of cases in which there is evidence of greater susceptibility to recurring ulceration, nor in treatment of recurring duodenal or gastrojejunal ulcer, for, in cases of recurring ulceration partial gastrectomy practically always will be followed by relative achlorhydria and recurrence of ulceration is exceedingly unlikely. It might appear that in cases of duodenal ulcer in which the preoperative gastric acidity is high, reduction of acidity might not be as great as in cases in which preoperative gastric acidity was lower and that patients whose preoperative gastric acidity was high would be prone to recurring ulceration after gastro-enterostomy. Nevertheless, in innumerable cases in which preoperative gastric acidity was high and the risk of removal of a large, penetrating and infiltrating duodenal ulcer did not seem warranted, I have performed gastro-enterostomy, with the consequence that maximal reduction of gastric acidity occurred (Cases 3, 4 and 5, Table II), even to the point of relative achlorhydria in some cases; moreover, the operations were followed by excellent clinical results.

COMMENT AND SUMMARY

Since reduction in gastric acidity occurs following any operative procedure which, as a result of anastomosis between the stomach and the duodenum or jejunum, allows reflux of intestinal secretion into the stomach, it appears that reduction in gastric acidity is largely attributable to the dilution or neutralization which occurs. Substantiating this conclusion is the fact that relative achlorhydria may occur after gastro-enterostomy as well as in the majority of cases in which resection of the stomach of the Pólya type is performed for duodenal ulcer, whereas loss of this factor of dilution, by performance of entero-anastomosis between the loops of jejunum, is followed by little reduction in gastric acidity.

Following pyloroplasty, or even gastric resection of the Billroth I type, in which the stomach is joined to the duodenum, the percentage of patients who have relative achlorhydria is not so great as when resection of the Pólya type, or gastro-enterostomy, are carried out and anastomosis is made between the jejunum and the stomach. This is probably attributable to the lack of reflux of duodenal secretion into the stomach because following the Billroth I operation, pyloroplasty or gastroduodenostomy the natural peristalsis carries the secretion onward. In contrast, following gastrojejunal anastomosis the peristaltic force carries jejunal secretion into the stomach. It is true that in the Billroth I type of resection and anastomosis as large a portion of stomach usually is not removed as is resected in the Pólya operation; this may or may not be a factor in the proportional reduction of gastric acidity. The response in acidity, to gastric resection for gastric ulcer, whether of the Billroth I or

the Pólya type, is practically universally constant and relative achlorhydria occurs practically without exception. In cases of gastric ulcers in which for various reasons, gastric resection is inadvisable, not infrequently relative achlorhydria will develop after excision of the ulcer when excision is combined with gastro-enterostomy; also interestingly enough, in a few instances when only the gastric ulcer is excised.

Gastric resection is the operation of choice in most cases of accessible, large, callous gastric ulcer and has its place in selected cases of duodenal ulcer, particularly when the ulcer is of the hemorrhagic type, as well as in cases of recurring duodenal ulcer and gastrojejunal ulcer. Furthermore, as time passes, other evidence may present itself, further indicating the value of gastric resection in cases of duodenal ulcer. However, for the present the arguments offered to show that it is better than other procedures are not convincing. These arguments are that in gastric resection the areas of gastritis associated with most ulcers are removed, that relative achlorhydria occurs after gastric resection and that this relative achlorhydria prevents recurring ulceration. However, in my experience gastritis has not been associated with most ulcers; moreover, relative achlorhydria follows gastric resection in only about 50 per cent of cases. And once again, the routine use of an operation which carries an increased risk and which may produce some undesirable physiologic disturbances, seems unwarranted.

A report has come from one clinic concerning 108 cases in which partial or subtotal gastrectomy had been performed for duodenal ulcer. In 47 of these cases relative achlorhydria failed to develop and in nine of the 47 gastrojejunal ulcer developed. This would indicate that among certain groups of patients in certain areas of the United States primary gastric resection for duodenal ulcer may be followed by a greater incidence of recurring ulceration than that which follows the conservative operative procedures of gastroenterostomy and pyloroplasty or gastroduodenostomy when applied to other groups of patients in other parts of the country.

Lastly, in the evaluation of any operation "the factor of geomedical variation of types of lesions and the part played by racial factors in these differences is as yet unknown to science."[1]

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A TECHNIC FOR THE MANAGEMENT OF GASTROJEJUNAL ULCERS WITH OR WITHOUT GASTROCOLIC OR JEJUNOCOLIC FISTULA

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THE technic suggested in this paper has been employed in only five instances. The report therefore is not made on evidence of its proved value, but rather on the theoretical conceptions of its original planning supported by such success as these few cases show.

The thesis is based on certain observations made years ago for a totally different purpose; some of the work of Mann and others on the experimental production and healing of chronic duodenal ulcers; and a determination never again to attempt an operative procedure of the severity carried out in the first patient of this series.

Jejunal ulcers are reported in widely differing percentages by different writers, but from whatever source one draws, they do occur, though perhaps not as frequently as they did when gastro-enterostomy was performed with less discrimination than is now generally practiced.

Jejunal ulcers vary in extent from a small marginal ulcer which offers little or no difficulty to the undoing of a previous gastro-enterostomy, to those extensive ulcers which have destroyed the jejunal wall, penetrated into the thickened mesocolon and are so firmly attached to the under surface of the colon that they cannot be separated from it. The ulcer may have actually formed a gastrocolic fistula. It is for these severer grades of extension that the procedure is designed.

Resection of the jejunum, transverse colon and stomach has been successfully performed in a few instances, but must always be a formidable undertaking, involving a grave risk to life, only to be accepted if no simpler measure can be employed. There have been not a few attempts made to offer means of avoiding such an extensive resection. This is another presented for comment and criticism. I cannot state that I have gone over with certainty all the descriptions of operations designed for the same purpose; but I can say that I have read all the references easily available and have found only one reference which suggested a somewhat similar procedure, reported by Andrews; even here, however, an essential part of the method suggested was not mentioned, namely, the deliberate use of the denuded cuffs to close the stoma without in any way encroaching upon its lumen.

The fate of the first patient in this series emphasized to the writer the necessity for some simpler operative measure than was there adopted. The patient was a man 49 years old, who, five years before, had had a gastro-enterostomy performed upon him for a chronic duodenal ulcer. There was

at the time of gastro-enterostomy a marked delay in emptying the stomach. The gastro-enterostomy had relieved his symptoms for four years, after which he began to experience pain in the epigastrium, loss of appetite, occasional vomiting. The pain was intolerable. The roentgenologic diagnosis was jejunal ulcer.

At operation there was found a mass at the stoma formed by a large ulcer which had destroyed the jejunum opposite the opening. The mass was so firmly fixed to the transverse colon that it could not be separated. An attempt to separate the two resulted in a small opening into the transverse colon.

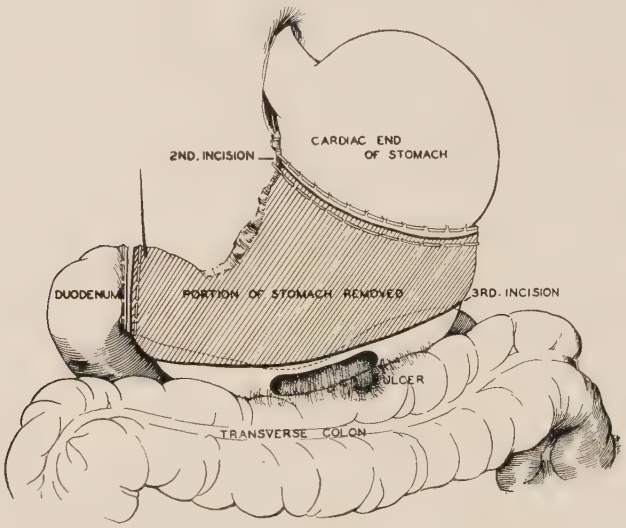

FIG. 1.—Showing the portion of the stomach which will be ultimately removed and the stoma unfortunately not where one could be placed. Its shaded portion is the part of the stomach to be removed. The unshaded portion of the stomach represents the cuff to be left attached to the jejunum above the transverse colon.

It was felt at the time that there was no alternative but resection and anastomosis of the jejunum, transverse colon and stomach. This was effected, and it seemed successful for seven days, when a jejunal fistula developed and he died 14 days after operation.

Years ago, when working with cats on another problem which required the opening and closing of the duodenum, it was found, following a suggestion by Doctor Archibald, that it was possible to get a satisfactory union by scraping away the mucosa from the edges of the bowel incision and then suturing the everted edges instead of the usual inturning as employed in the suture of intestine in the usual anastomosis in the human.

At a later period the same principle was used in resection of the stomach, when for any reason it was desired to cut the stomach cross proximal to the pylorus. Under the circumstances, it makes a clumsy and perhaps unsafe closure to turn the stomach in, as is usually done. The remnant of the

pyloric end of the stomach instead of being inverted was opened, the mucosa dissected off the muscularis down to the junction with the duodenal mucosa. The muscular walls were then sutured together, everting the edges and using no serosal suture. This makes a perfectly satisfactory closure in a section of the stomach just proximal to the pyloric opening.

Experiments by Mann and others have shown that chronic ulcers can be produced by exposing the duodenal mucosa to the gastric secretions unmodified by the normal duodenal contents; further, that these experimental ulcers ordinarily heal if the relationships are restored; if, in other words, the duodenal mucosa is once again protected by its normal alkaline secretion. It has been the experience of surgeons that duodenal ulcers, which cannot be excised, commonly heal when shut off from the gastric juices by one of the operations involving pyloric exclusion.

It was thought that if the jejunal ulcer could be shut away from the gastric contents it would also heal. If at the same time the exclusion of the

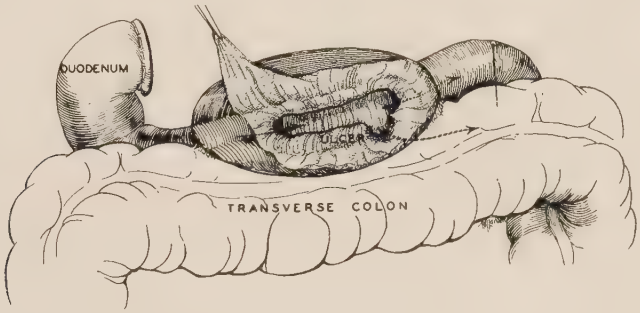

FIG. 2.—Represents the dissection of the gastric mucosa down to the edge of the ulcer. This dissection is easily made because of the looseness of the mucosa of the stomach on the muscularis.

jejunal ulcer could be accomplished without encroaching on the ulcer itself, there would be no need to resect the jejunum and the transverse colon where the ulcer had extended into its wall.

The two requirements, namely, the exclusion of the jejunal ulcer from the acid secretions of the stomach and the closure of the stoma without encroaching on the jejunum, it was thought, could be met by making a wide cuff from the stomach proximal to the stoma. The stomach mucosa could then be carefully dissected away from the inner surface of the cuff, down to the muscularis and right up to the edge of the ulcer. These two broad surfaces could then be sutured together, muscularis to muscularis. In this way the stoma would be closed, the jejunal ulcer and transverse colon left untouched and the ulcer protected from the acid gastric juices. It should heal as does the experimental ulcer and the excluded duodenal ulcer. The stomach could then be resected and the operation concluded by an anterior gastrojejunal anastomosis with or without an entero-anastomosis (Figs. 1, 2 and 3).

Not long aferwards, a patient presented himself with a condition closely resembling that first cited, namely, a definite history of duodenal ulcer for which a gastro-enterostomy had been performed three years before. He was suffering pain, occasionally vomited, the stool showed blood in small quantities; he was losing weight. Roentgenologic examination confirmed the diagnosis of jejunal ulcer. It was decided to operate and apply the theoretically evolved procedure.

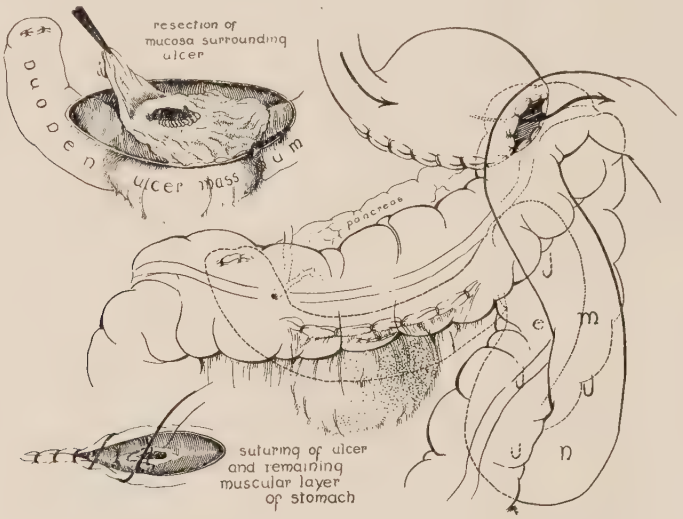

FIG. 3.—Is a composite diagram representing various stages of the closure and the suture of the surfaces of the denuded stomach cuff.

CASE REPORTS

Case 1.—Mr. P. 84711. Age 49. Chief Complaint.—Pain in epigastrium, blood in the stools. History.—There has been a vague abdominal history for ten years. One year ago he was operated upon for a duodenal ulcer, and a posterior no loop gastro-enterostomy was performed. Three months ago pain recurred and roentgenologic examination revealed a jejunal ulcer as indicated by a "rest" after the barium meal.

Operation.—April 12, 1933. A large mass of inflammatory tissue was found in the neighborhood of the gastro-enterostomy opening. It was two and one-half inches long and two inches in diameter, and situated immediately under the transverse colon. The transverse colon itself was edematous and thickened. The stomach was dissected free; cut across as if for a three-quarters resection. In this way it was entirely freed, except for its attachment to the jejunum. A cuff of stomach was then cut one and one-half inches from the edge of the stoma. The mucosa was carefully dissected off the muscularis down to the edge of the ulcer until all the stomach mucosa was removed.

It could then be seen that there was a large ulcer in the jejunum at least one and one-half inches in diameter, of which one wall must have been formed by the transverse colon. The stoma was closed by suturing together the inner surfaces of the stomach cuff, and the operation completed by an anterior gastrojejunostomy, to the remnant of the stomach.

The patient was immediately relieved of his pain and during the subsequent three months gained 30 pounds. He was well until the autumn of 1935, when he returned, complaining of pain. Roentgenograms suggest that he has a second jejunal ulcer at the site of the new opening.

Case 2.—Mrs. E. 88251. Age 60. Admitted February, 1934. Chief complaint.—Eructations of gas with a foul odor, loss of weight and strength. She had had a gastro-enterostomy in 1921, but was never free from symptoms. In July, 1933, she noticed pain and had eructations of foul smelling gas followed by diarrhea. A diagnosis of jejunal ulcer with gastrocolic fistula was confirmed by roentgenologic examination.

Operation.—February 24, 1934. The transverse colon was firmly attached to the jejunal opening and gas could be expelled from the colon into the stomach. The first step consisted in freeing the stomach from adhesions and tying the vessels along the greater and the lesser curvature. The duodenum was cut across and turned in, the stomach also as for a three-quarter resection. In this way it was freed except for an attachment to the jejunum. A cuff was then fashioned from the stomach; the mucosa dissected off to the edge of the ulcer and the stoma closed by suture of the two surfaces together, muscularis to muscularis. An anterior gastrojejunostomy completed the repair, leaving the ulcer and fistula intact.

Immediately following the operation she was relieved of pain and the eructations of foul gas ceased. She took nourishment and seemed on the way to recovery for four weeks, when she suddenly collapsed and died from peritonitis due to a leak from the corner of the anterior anastomosis.

At the autopsy the ulcer was shown not to be healed; the fistula had closed and the ulcer was definitely smaller than at operation.

Case 3.—J. P. Age 48. Admitted May, 1933. Chief Complaint.—Pain in the epigastrium not related to, or relieved by, food. Bleeding from the bowel. Operation five years before gastro-enterostomy for duodenal ulcer with obstruction. Recurrence of symptoms during last three months. Roentgenologic diagnosis of jejunal ulcer.

Operation.—May, 1933. Dense adhesions were found to the anterior abdominal wall. A large mass was present representing an ulcer of the jejunum opposite the stoma and firmly adherent to the transverse colon. The stomach was freed, and resected. A cuff was then fashioned from the stomach, the mucosa dissected off and the stoma closed by suturing the opposed surfaces of the stomach wall one to the other. The operation was completed by an anterior gastrojejunostomy.

The patient was relieved of his pain, bleeding stopped and he has been fairly well since. He had occasional distress for three to four months, after which he could eat fairly freely without pain.

Case 4.—M. J. Age 37. Admitted May 3, 1933. Chief complaint.—Pain in abdomen, vomiting and tenderness in epigastrium. He had been operated upon in 1923 and a gastrojejunostomy performed. He was fairly well for nine years, but was readmitted in 1932 for pain. Roentgenologic diagnosis of jejunal ulcer.

Operation.—June 16, 1933. Many adhesions were found attaching the stomach to the anterior wall. The transverse colon was firmly attached to the stomach near the stoma as was the jejunum to the colon. There was a small mass felt in the region of the stoma.

The stomach was gradually freed from adhesions till the pylorus and duodenum were prepared for resection. The stomach was cut across one inch proximal to the pylorus, and the pylorus closed by dissecting off the gastric mucosa and suturing the muscle surfaces together. The stomach was then cut across as for a resection. The main portion of the stomach was then cut away from the stoma, leaving a fringe or cuff of stomach wall about one and one-half inches wide. The gastric mucosa was dissected off down to the stoma and the stoma closed by suturing the bared surfaces together; and the repair completed by an anterior gastro-enterostomy.

This patient might have been dealt with by other methods, but this seemed to the writer to be easy and has had a good result. He has gained 15 to 16 pounds; no pain, no vomiting, no acidity. He is working and taking ordinary food, other than fried or spiced.

Case 5.—H. B. Age 52. Admitted September, 1932. Chief complaint.—Pain in the abdomen, loss of weight, tenderness and pallor. He had been operated upon for gastric

ulcer in 1927. Symptoms recurred in 1932. Roentgenologic diagnosis, doubtful jejunal ulcer.

Operation.—September 20, 1932. A hard mass was found in the region of the gastro-enterostomy opening. After dissection this was found to be an ulcer more on the stomach than the jejunal side of the anastomosis. As in other cases, the stomach was dissected free, the stoma closed by making a cuff from the stomach wall and closure effected by suturing these surfaces together after dissecting off the mucosa. The final anastomosis was made posteriorly, because the mesentery of the small intestine was too short.

This patient was discharged in 28 days, feeling well, but still on a restricted diet; he had no pain or vomiting and was gaining weight.

The principles here made use of seem so evident that they must often have been used; but I have read with some degree of care several of the larger bibliographies and have not, as it seemed to me, found them so clearly enunciated as to form the basis of a planned method of treatment. If this is an old story, then these cases are offered as additional evidence of its usefulness. If there is anything new, it may be found worthy of your consideration.

Discussion.—Dr. William L. Estes, Jr. (Bethlehem, Pa.).—I have been very much interested in Doctor Scrimger's technic, because four years ago we reported a somewhat similar procedure before the Johns Hopkins Surgical Society. It was a patient upon whom a posterior gastro-enterostomy had been performed in conjunction with the closure of a perforated ulcer, seven years before. He had also had a perforated jejunal ulcer two years before he came to us for treatment. Examination disclosed evidences of marked obstruction in and about the gastrojejunal area, resulting from a large ulcer. He also had been having repeated hematemeses and evidences of gastric retention.

At operation we found a hugely dilated stomach above a very extensive gastrojejunal ulcer, the type Doctor Scrimger has described, with its base very close to the transverse colon—intimating that there might shortly have developed a gastrocolic or jejunocolic fistula. There were present a dilated proximal jejunum and a rather marked obstruction in what was the distal loop of the previous gastro-enterostomy, the site of the former perforated jejunal ulcer.

Primarily, we had a very bad risk patient to deal with. Therefore, our first procedure, similar to the first stage of Doctor Scrimger's operation, was to expose the gastrojejunal ulcer from above through an opening in the gastrocolic omentum. The stomach was easily separated from the upper margin of the stoma. The opening into it was closed by a double layer of sutures. The upper margin of the stoma, without disturbing the mucosa, was turned in and firmly closed by two layers of sutures. The operation was concluded by making an anastomosis between the distal and proximal loops of jejunum, thus side-tracking the ulcer.

The remarkable thing was not the fact that he did quite well after the operation, but that he returned after nine weeks with evidences of reactivation of his old duodenal ulcer. At reoperation we found that there had been complete healing of the gastrojejunal ulcer area. There was no further induration in the mesentery of the transverse colon, and there was a marked atrophy of the side-tracked loop of the jejunum.

I therefore believe that this, or an operation similar to the one Doctor

Scrimger reported, has a place in the treatment of these very advanced cases of gastrojejunal ulcer.

Dr. Frank H. Lahey (Boston, Mass.).—May we ask Doctor Scrimger how he avoids regurgitant fecal contamination. I presume he must have some plan.

Dr. Francis A. C. Scrimger (Montreal, Canada) closing.—I have nothing to add except to indicate that my suspicions were correct, and that the ideas contained in this had been already recognized, although my diligence had not been sufficient to discover them all, and to point out that it seemed to me an advantage that the removal of the gastric mucosa was perhaps a principle worthy of some consideration.

Relative to Doctor Lahey's query concerning contamination, I have no plan for that, because in only one case was the fistula present, and it was quite small, and while there had been regurgitant contamination into the stomach, at the time the bowel did not regurgitate.

EXPERIMENTAL LYMPHEDEMA OF THE INTESTINAL TRACT AND ITS RELATION TO REGIONAL CICATRIZING ENTERITIS*

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THE anatomic, physiologic and embryologic studies of the lymphatics during the past 300 years have firmly established them as a definite part of the vascular system. The clinical importance of this system of vessels and nodes has only recently been realized as new methods have been developed for the study of its physiologic functions and pathologic alterations.

Investigators learned, as had been ascertained previously for the blood vascular system, that the lymphatics grew into new tissues, that they regenerated and developed a collateral circulation, that they became inflamed, sclerosed and thrombosed. Recently it was found that lymphatics could be obliterated in an extremity resulting in a chronic lymphedema or elephantiasis. This same method may be applied to the lymphatics of other parts of the body and to various organs. We became interested in the effect of obliteration of the intestinal lymphatics in localized portions of the bowel after observing somewhat unusual lesions in clinical cases in which the appearance of the bowel, after the possibility of malignancy was eliminated, suggested a chronic lymphatic obstruction.

Resected bowel from three such cases, seen on the surgical service, was classified as cicatrizing regional enteritis and its pathologic study revealed a marked edema and fibrosis of the wall and engorgement of the lymphatics. We felt this picture resembled in many details that of chronic lymphedema or elephantiasis, as seen in the extremities. The two dominant features of the intestinal lesion seemed to be a low grade chronic infection with a concomitant chronic lymphatic obstruction and edema.

Fortunately for us the work of Drinker, Fields and Homans[1] on the experimental production of lymphedema and elephantiasis in the limb of dogs had been just published. Some years ago one of us,[2] at the suggestion of the late Professor Halsted, had attempted to produce elephantiasis in animals but the work reported by Homans, Drinker and Fields[3] presented a satisfactory experimental method of obtaining chronic lymphedema which we have applied in the study of this condition in the intestinal tract of dogs.

METHOD.—Drinker and Fields developed an elaborate method of tying a fine quartz cannula into the lymphatic vessel through which a 2½ per cent solution of quinine hydrochloride combined with a suspension of crystalline silica dust was slowly injected. By repeated injections of the irritating and

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sclerosing materials the lymphatic vessels and nodes were injured and became obliterated by a fibrosis, producing the condition of chronic lymphedema.

During previous experimental work on the lymphatics,[4] under Doctor Sabin, a technic for lymphatic injection was developed, using fine hypodermic needles, gauge 27 and 28, attached to a tuberculin syringe. We found this method well adapted for the direct injection of the mesenteric lymphatics as well as of the subserosal lymphatics of the bowel, without interfering in any way with the intestinal blood vascular network, thereby allowing the study of the isolated effect of lymphatic occlusion in the intestinal tract and the end-results of lymphatic fibrosis produced by irritating and sclerosing materials. Adjacent bowel with undisturbed lymphatic drainage to uninvolved lymph nodes was used as a control in these studies.

The material used for lymphatic injections included crystalline silica (200) mesh, Hill's bismuth oxychloride mass, as modified by Poth[5] for lymphatic injection, rose aniline dye in the form of indelible lead, or sodium morrhuate added to the bismuth mass as a sclerosing material. In some instances appendiceal contents were added to the bismuth mixtures before injection and in others a suspension from 24 hour broth culture of *B. coli* was given intravenously one to three hours before the lymphatic injections. One, two or three reinjections were made in a number of animals at intervals from several weeks to months after the preceding treatment.

Subserosal lymphatic injections in the stomach and pylorus were difficult to effect, but subserosal and mesenteric lymphatics could be injected readily in the duodenum, distal ileum and proximal large bowel. The mesenteric lymphatic vessels of the ileum were filled easily and, at times, these were the only injections made at the first operation, with mesenteric and subserosal injections made at subsequent explorations (Fig. 1). The present report will be confined to the results of lymphatic obstruction in the ileum and colon.

A paralysis or dilatation of the intestinal lymphatics was noted when intravenous bacteria had been given an hour before operation, and the lymphatic dilatation was particularly remarkable in three animals made sensitive to foreign protein by seven intravenous injections of horse serum during the previous six weeks. In these animals a final injection of serum was given just before operation. Evidence of anaphylactic shock was apparent by the cold skin and the cold intestines, which were pale, relaxed and showed no peristalsis on irritation. The arteries and veins were contracted, but the lymphatics and lacteals were found to be dilated or paralyzed, were easily injected and appeared as large vessels in the mesentery, subserosa and between the muscle layers.

The experiments were concluded by sacrificing the animals under ether anesthesia. The most satisfactory procedure for fixation was to remove the whole gastro-intestinal tract, to separate any adhesions or matted loops of bowel under warm saline and then to fill partially the lumen with warm saline. When all of the intestines were completely relaxed they were quickly put into formalin, thus insuring simultaneous contraction and fixation of all

parts of the tract. Areas of the treated and normal bowel were excised later for microscopic study.

Experimental Results.—No free peritoneal fluid was ever seen upon opening the abdomen for subsequent reinjection, or when the animal was sacrificed, but adhesions about the injected mesentery and between the omentum and loops of treated bowel were encountered frequently. The treated

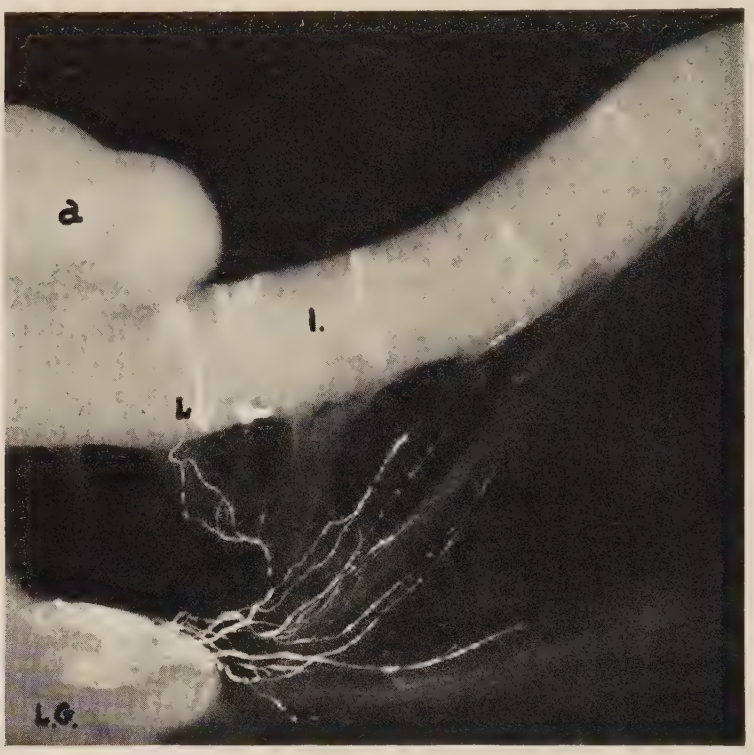

Fig. 1.—Roentgenogram of normal ileum and mesentery in which the lymphatics were injected directly with bismuth oxychloride (26 per cent) in an acute experiment. (a) Tip of appendix. (I) Ileum. (L.G.) Regional lymph node filled partially with bismuth from mesenteric lymphatic and subserosal lymphatic injections. (L) Lacteal filled from subserosal injection.

segment felt thickened, and some of the mesenteric lymphatics were dilated while others were sclerosed. The regional lymph nodes were enlarged and firm, and the involved mesentery frequently scarred. In only one animal did the injections fail to produce chronic pathologic changes persisting for months to a year.

Specimens were studied from 19 dogs in which there were intestinal lymphatic injections. Bismuth oxychloride (26 per cent) alone was used in three injections, bismuth oxychloride and sodium morrhuate (5 per cent), equal parts, were used in 15 injections, while the mixture of bismuth and rose aniline dye was employed seven times. Silica and rose aniline dye were used only twice because the silica interfered with the smooth movement of the plunger of the syringe. Bacteria mixed with the bismuth and rose aniline

dye were used twice, and intravenous injections of *B. coli*, followed in one to three hours by lymphatic injections with bismuth and the dye or sodium morrhuate, were used four times.

Acute Lymphedema.—Six animals died or were sacrificed at intervals

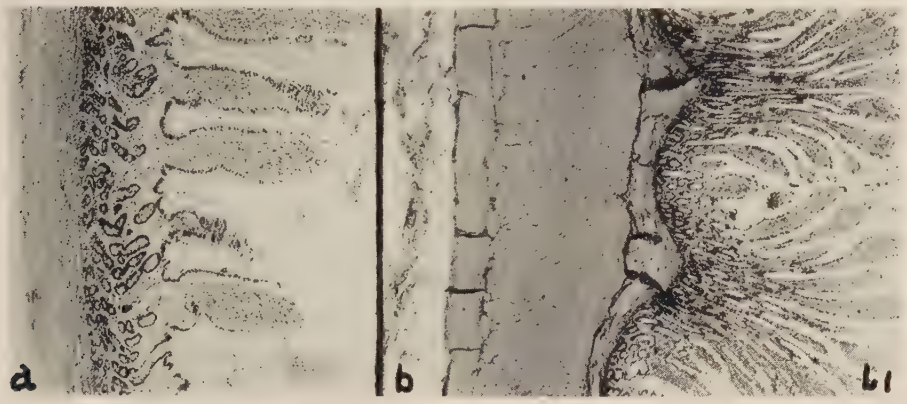

Fig. 2.—L 1. Four and one-half week specimen. (a) Normal ileum (×34). (b) Ileum (×34), mesenteric lymphatics injected with bismuth oxychloride, showing muscle layers edematous and thickened four to five times more than normal. Lacteals engorged. Submucosa thickened and infiltrated.

from a few days to two weeks. The histologic picture varied with the length of survival. The thickened bowel wall showed inflammation and thickening of the serosa and edema of the circular and longitudinal muscles, with the lacteals in the intermuscular septum distended with fluid and lymphocytes.

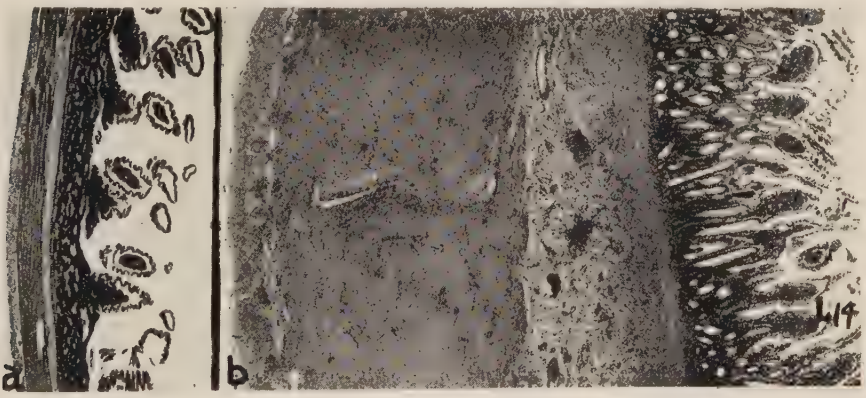

Fig. 3.—L 14. Nine and one-half week specimen. (a) Normal ileum (×20). (b) Ileum (×20). Intravenous injection of *B.coli*, followed in three hours by mesenteric and subserosal ileac lymphatic injections of bismuth and rose aniline dye. Inner circular and outer longitudinal muscles edematous and eight times normal thickness. Lacteals engorged and thrombosed. Submucosa greatly infiltrated, 15 times normal thickness.

The submucosa was swollen and infiltrated with leukocytes and its lymphatics were engorged with cells. The mucosa seemed a little swollen but there were no ulcerations.

Between two and four weeks after injection the lymphatics were noticeably

filled with very large pale cells containing large pale nuclei, and the vessels seemed thrombosed and sclerosed, so that a chronic stage had developed.

Chronic Lymphedema.—In 13 animals the lymphatic injections had been

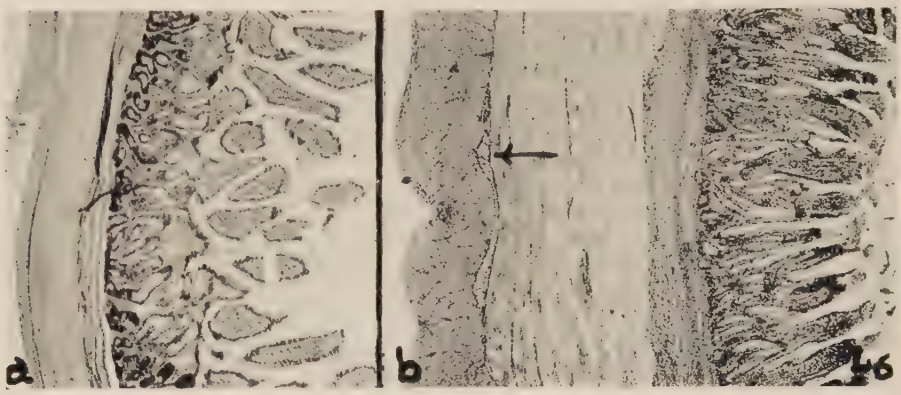

Fig. 4.—L 6. Twelve month specimen. (a) Normal ileum (×18). (b) Ileum (×18). Twelve and ten months previously, ileal mesenteric and subserosal lymphatics injected with equal parts of 26 per cent bismuth oxychloride and 5 per cent sodium morrhuate. Serosa thickened and infiltrated. Muscles edematous and four times normal thickness. Arrow indicates an engorged and thrombosed lacteal (Fig. 5). Submucosa thickened by edema, lymphocytic infiltration and fibroblasts. Lymphatics engorged and thrombosed.

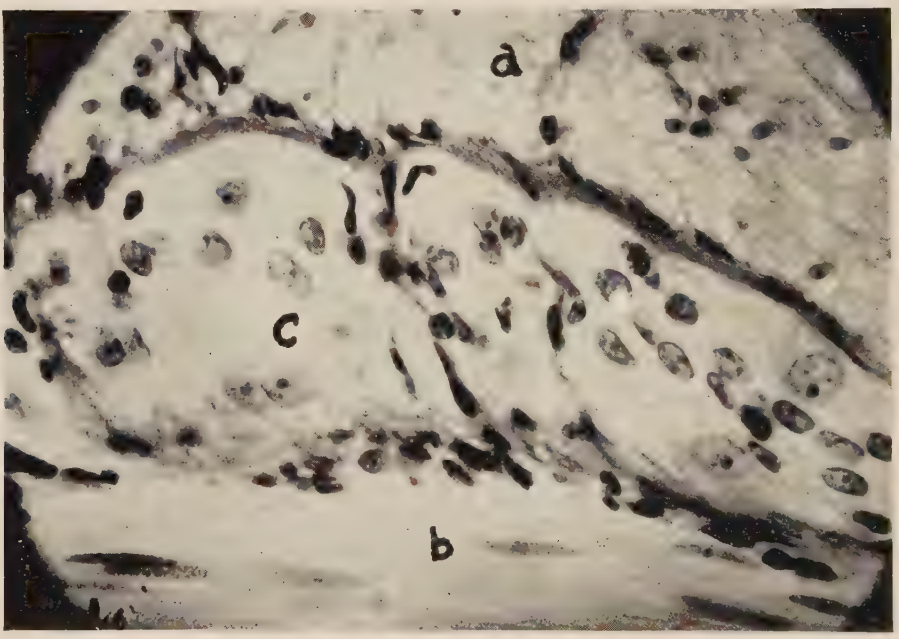

Fig. 5.—L 6. High power (×650), of intermuscular thrombosed lacteal (c), engorged with large pale mononuclear cells. The adjacent outer longitudinal (a) and inner circular (b) muscles are pale and edematous.

made one month or longer before death and all except one showed definite gross and microscopic alterations in the involved bowel. Of the 12 dogs that had developed a chronic lymphedema three were injected only once, six

were injected twice, two were injected three times and one was injected four times. Yet all had the same characteristic histologic changes.

That chronic pathologic changes which persisted, without subsidence, for months should develop after even a single injection of irritating and sclerosing substances into the intestinal lymphatics was striking and unexpected.

The main differences between the acute and the chronic changes following the injections were found to be in the lymphatics and in the submucosal and muscular layers. With subsidence of the acute phase the signs of inflammation disappeared, and only round cells and lymphocytes were to be seen. The edema of the tissues persisted, since the submucosal lymphatics and the lacteals lying between the muscular layers had been obliterated by sclerosis or thrombosis, and were now filled with firm trabeculations and packed with very large, pale cells containing large, lightly staining nuclei (Fig. 5). The greatest change was a thickening of the longitudinal and circular muscles and

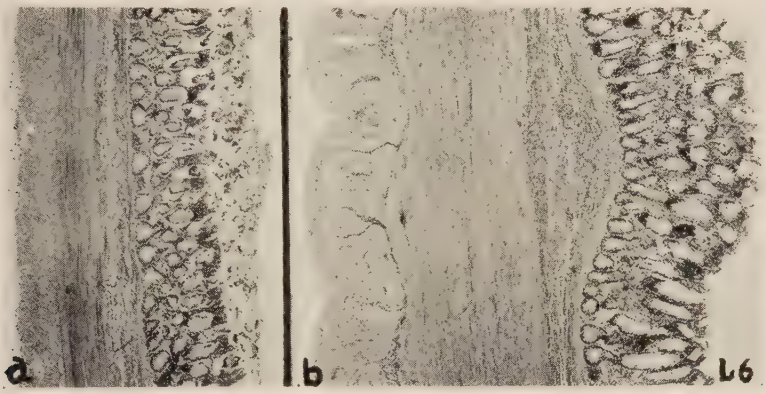

FIG. 6.—L 6. Twelve month specimen. (a) Normal colon (×24). (b) Colon (×24). Twelve and ten months previously subserosal lymphatics injected with equal parts of 26 per cent bismuth oxychloride and 5 per cent sodium morrhuate. Serosa thickened and fibrinous. Muscles edematous and two to three times normal thickness, with lacteals engorged and thrombosed. Submucosa thickened, infiltrated and fibrosed.

in the submucosa, these being two to eight times thicker than normal; and due to the chronic edema present the muscle fibers were found to be swollen and indistinct (Figs. 2, 3, 4, 5 and 6). Edema, fibrin and fibrosis persisted in the submucosa, and usually the serosa was somewhat thickened (Figs. 4 and 6). No ulcerations of the mucosa were seen.

These pathologic changes were seen in all the 12 specimens, and no appreciable differences were found following injections of bismuth in conjunction with sodium morrhuate (Figs. 4, 5 and 6) or rose aniline dye, and of silica in conjunction with the aniline dye. Bismuth alone (Fig. 2) produced definite alterations, but the most marked chronic lymphedema occurred when *B. coli* was given intravenously shortly before the lymphatic injections of bismuth and rose aniline dye (Fig. 3).

The end-result of sclerosing and obliterating the lymphatics to a segment of intestine was to produce a bowel thickened from chronic lymph stasis with the lymphedema most marked in the submucosal and muscular layers. This

characteristic pathologic picture (Figs. 2, 3, 4 and 6) was found in 12 specimens, although the number of injections ranged from one to four, although several different sclerosing materials had been used and although one to ten months had elapsed between the injections and the autopsy.

PROTOCOLS

Since the number and location of the lymphatic injections, the substances used for lymphatic sclerosis, and the gross pathologic observations at reoperation or autopsy have been summarized, it will not be necessary to give the details in all of the experiments. The reports of three experiments, with photomicrographs, are presented briefly to illustrate the findings in chronic lymphedema of the ileum and colon.

L-1.—On October 24, 1934, the mesenteric lymphatics were directly injected close to the ileum with 26 per cent bismuth oxychloride. The white bismuth filled these lymphatics and finally filled the regional lymph node, draining about 12 to 15 inches of the terminal ileum. No subserosal lymphatic injection was made.

November 11, 1934. Three weeks later an exploration was made. The ileum was bound down by adhesions, and the injected lymph node was quite large. One distended ileac mesenteric lymphatic was reinjected.

On November 26, 1934, four and one-half weeks after the first injection, the dog was sacrificed. No free fluid or distended loops of bowel were seen. A large mass of matted loops of small bowel in the cecal region with shortened and thickened mesentery was dissected free. A large ileal lymph node was surrounded by fibrous adhesions. The intestinal tract was partially distended and fixed in formalin for one hour, then incised longitudinally at the antimesenteric border and wrapped loosely on a plate of glass for further fixation. The terminal six inches of ileum were thickened but no mucosal ulcerations were seen.

Microscopic Findings.—The section from the injected ileum (Fig. 2 b) showed the serosa very thick and infiltrated.

The inner circular and outer longitudinal muscles were four to five times thicker than the normal ileum (Fig. 2 a) and were edematous, with slight round cell infiltration and with the lacteals engorged with so called plasma cells and very large, pale cells. The submucosa was thickened and infiltrated with engorged lymphatics. The mucosa was intact.

L-14.—On August 1, 1935, at 12:30 P.M., three-fourths of a cubic centimeter of a 24 hour broth culture of *B. coli* was given intravenously. At 2:30 P.M., five ileal mesenteric lymphatics were injected with bismuth oxychloride, which was colored a deep purple by adding a piece of indelible lead (rose aniline dye). Four subserosal lymphatics were injected in the ileum, two in the appendix and one in the proximal colon three inches from the ileocecal valve.

On October 7, 1935, nine and one-half weeks after injection, the animal was sacrificed. No free fluid or adhesions were seen. Definite thickening of the terminal ileum was felt. The intestinal tract was removed, partially filled with warm saline and, when entirely relaxed, put into formalin.

Microscopic Findings.—Section of the injected ileum (Fig. 3 b) showed the serosa edematous and slightly infiltrated. The muscles, especially the inner circular, were markedly thickened to eight times that of the normal ileum (Fig. 3 a) and showed edema and slight round cell infiltration with engorged and thrombosed lacteals. The submucosa was greatly infiltrated with dense areas of round cells and engorged lymphatics, and was 15 times thicker than in the control section. The mucosa was intact. The nor-

mal ileum (Fig. 3 a) showed a single layer of serosal cells with the muscle fibers distinct and the lacteals containing only lymph. In the submucosa the lymphatics were small.

L-6.—On December 13, 1934, the distal 12 inches of the ileal mesentery had the lymphatics injected, and four subserosal ileal lymphatics were also injected with equal parts of bismuth oxychloride (26 per cent) and sodium morrhuate (5 per cent). Subserosal lymphatic injections were also made on the proximal three inches of large bowel.

On January 28, 1935, six and one-half weeks later, reinjection of lymphatics was done. Only a few omental adhesions were seen. The ileum was thickened. Many subserosal lymphatics in the distal ten inches of ileum were readily injected and filled the deeper enlarged lacteals. The colon was thickened. Three subserosal lymphatics in the proximal colon were injected.

On November 7, 1935, nearly 12 months after the first, and ten months after the second injection, the animal was sacrificed. After mesenteric adhesions of the ileum were divided, definite thickening of ileum, appendix and proximal colon was felt.

Microscopic Findings.—In the sections of the injected ileum (Fig. 4 b), the serosa was thickened and contained dilated, thrombosed lymphatics. The inner circular muscle was edematous with the muscle fibers swollen and lacking detail (Fig. 5 b). Some wandering cells were seen. The longitudinal muscle showed the same edematous condition (Fig. 5 a). The muscle layers were four times thicker than in the normal ileum. Engorged lacteals (arrow in Fig. 4, and Fig. 5 c) were filled with large pale cells and showed thrombosis and, in some areas, cannulization. In the submucosa some round cell infiltration was seen just beneath the mucosa and there were frequent fibrin deposits and marked fibrosis causing this layer to be twice as thick as in the section of normal ileum (Fig. 4 a). Engorged and thrombosed lymphatics were numerous. The mucosa was intact. The section of the normal ileum (Fig. 4 a) showed the muscle fibers to be sharp and distended. The lacteals were not thrombosed, and they contained an occasional large pale cell. The serosa was composed of a single layer of cells.

In the section of the injected colon (Fig. 6 b) the findings were similar to those in the ileum with the serosa thickened and fibrinous. The muscles were two to three times thicker than normal, and were edematous. The lacteals were engorged and thrombosed. The submucosa was thickened, infiltrated and fibrosed. Five or six layers of lymphocytes were seen beneath the musoca, instead of two or three layers in the normal colon (Fig. 6 a).

Regional Cicatrizing Enteritis.—In these experiments the constant pathologic changes, grossly and microscopically, that became chronic and persisted for months after regional sclerosis and obstruction of the intestinal lymphatics to a portion of the ileum or colon, resembled in many respects the pathologic picture in clinical cases of regional cicatrizing enteritis. Many recent reports of cases showing this clinical entity have been published so that the symptoms, signs, and diagnosis of this disease need not be considered in this paper, although a short description of the pathologic findings obtained from the literature and studied in three of our clinical cases will be given.

When Crohn, Ginzburg and Oppenheimer,[6] in 1932, separated a subgroup from the benign, nonspecific granulomata and described regional enteritis as a pathologic and clinical entity, they stated that the etiology of the process was obscure. They felt that this disease of the terminal ileum was characterized by a subacute or chronic necrotizing and cicatrizing inflammation of all the coats of the ileum, which frequently led to stenosis of the lumen and was often associated later with fistulous formation and a palpable tumor in the right lower quadrant.

Etiology.—In the Stanford clinic, in September, 1933,[7] a unique and original observation was made in a case of presumed regional ileitis in a child aged six, all previous observations having been made in adults. In this patient, the lesion was completely excised, including the terminal ileum and four inches of the cecum. The mucosa of the ileum, appendix and colon was everywhere intact but characterized by an extensive diffuse edema which involved also the mesentery and regional lymph nodes. An extensive fibrosis accompanied this edema producing the localized mass. It was inferred from these observations in a child that the process was primarily a lymphadenitis and that ulceration of the mucosa, when present, was secondary to the lymphatic obstruction and infection.

Bell,[8] in 1934, reported that he was unable, by interference with the blood supply of the intestinal tract in animals, to produce a cicatrizing enteritis, ulceration of the mucosa or any lesion simulating this condition, and concluded that the pathologic process in this disease was not one of inadequate blood supply. Bell raised the question whether the etiologic factor was infection in the mucosa spreading to the intestinal wall with edema of the mesentery from inadequate lymphatic drainage, or whether it was an infection starting in the lymphatics as a lymphangitis, and extending to the wall of the bowel.

Bockus and Lee[9] stated that any primary inflammatory mucosal disease in the terminal ileum might in the end resemble the entity of regional ileitis. Ginzburg and Oppenheimer,[10] for instance, suggested a disturbance in the vascular mechanism, as found in self-reducing intussusception or recurrent partial volvulus, as a possible rôle in this disease, and Homans and Haas,[11] Erb and Farmer,[12] and others suggest primary appendiceal disease as an etiologic agent with secondary involvement of the mesentery and, finally, the terminal ileum. Bockus and Lee[9] conceive the possibility of a lymphangitis and surrounding inflammation which might encroach upon or infect the blood supply, producing a slow devitalization of the terminal segment of ileum, resulting in terminal ileitis. Felsen[13] offers evidence that a great majority of cases of chronic, nonspecific, ulcerative colitis and ileitis, and nonspecific granuloma are the chronic stages of acute bacillary dysentery.

Clinical Pathologic Reports.—The description of the clinical entity involving the terminal ileum was made in 1932, but subsequent reports have been made of similar pathologic conditions which involve the jejunum, ileum, cecum and colon, so that the term chronic cicatrizing enteritis, as suggested by Harris, Bell and Brunn,[14] is more comprehensive.

Crohn and his associates studied specimens obtained from patients who had been ill for at least a year. Ulceration of the mucosa with blunting of the villi from edema, marked inflammatory, hyperplastic and exudative changes in the submucosal and muscular layers, and thickened, fibrotic serosa produced an enormously thickened intestinal wall which encroached upon the lumen. The mesentery was greatly thickened and fibrotic. Histologically, various degrees of acute, subacute and chronic inflammation were shown by

the presence of polymorphonuclear, round cell, plasma cell and fibroblastic elements. In some cases the presence of giant cells was striking. Near them were inclusion vegetable cells which had apparently resulted in the formation of the giant cells. After careful study they found no evidence of tuberculosis, syphilis, actinomycosis, Hodgkins' disease or lymphosarcoma.

An important contribution to the clinical study of this disease was the presentation of an early stage as reported by Erb and Farmer,[12] who gave an account of four children with acute ileocolitis simulating appendicitis and characterized by edema of the ileocecal region and mesenteric nodes. They felt that the disease they described was closely related to regional ileitis, or chronic cicatrizing enteritis or benign granuloma of the intestines. The pathologic finding in their fourth case—of a child two and one-half years old, ill for six days—was almost identical with the findings observed in the acute cases described by Crohn[15] where the terminal ileum was found to be thickened, soggy, and edematous, the serosa a blotchy red and the mesentery greatly thickened and containing numerous hyperplastic nodes. Histologically the mucosa was ulcerated and covered with a thick layer of exudate. The lymphoid tissue had undergone marked necrosis. Extensive fibrin formation was seen, and many large mononuclear cells interpreted as endothelial leukocytes were present in the intestinal wall. They noted a sparsity of polymorphonuclear cells. The edema led to a marked increase in thickness of the bowel wall and involved the muscular layers and particularly the submucosa.

CASE REPORTS

Case 1.—It was this specimen of chronic ileitis (Fig. 7) resected by Dr. G. Nagel from a man (E. D.), aged 27, that suggested to us a picture resembling chronic lymphedema and led to the experimental production of chronic intestinal lymphedema herein reported. In the photograph a match stick indicates the sinus from the cecum that had persisted for two years subsequent to an appendectomy.

The greatly thickened terminal ileum when cut showed the serosa thickened by areolar and fibrous tissue which had a heavy perivascular infiltration of round cells (Fig. 8). The muscles were greatly thickened, edematous and the inner circular muscle layer was heavily infiltrated with fibroblast and round cells adjacent to the submucosa, which likewise showed marked fibrosis and infiltration. The thrombosed lacteals were engorged with the large pale cells and the submucosal lymphatics were thrombosed and distended with the same large mononuclear cells. The mucosa was ulcerated in places, and elsewhere it was heavily infiltrated with lymphocytes, and leukocytes. Lymphoid tissue and clusters of lymphocytes were frequently seen beneath the mucosa and the serosa.

Case 2.—In Holman's[7] discussion of Bell's[8] paper, he reported the boy, aged six (C. L.), who had symptoms of appendicitis for only one day, but in the preceding two years had had occasional abdominal pain. At operation the expected appendiceal abscess was found to be a firm mass, involving the terminal ileum, cecum and base of the appendix which was resected. The mesentery was short and thick, and had enlarged lymph nodes.

Our pathologic study of the tissue showed the terminal ileum and appendix to be edematous and thickened with the ileal mucosa studded with lymphoid masses and numerous petechial hemorrhages but no ulceration. The submucosa was thickened and showed thrombosed lymphatics and scattered round cell infiltration. The muscle layers

were greatly thickened and distorted, showed moderate lymphocytic infiltration and their edematous condition was seen by the indistinct outlines of the muscle fibers (Fig. 91). The lacteals were engorged with large pale cells which some writers have described as

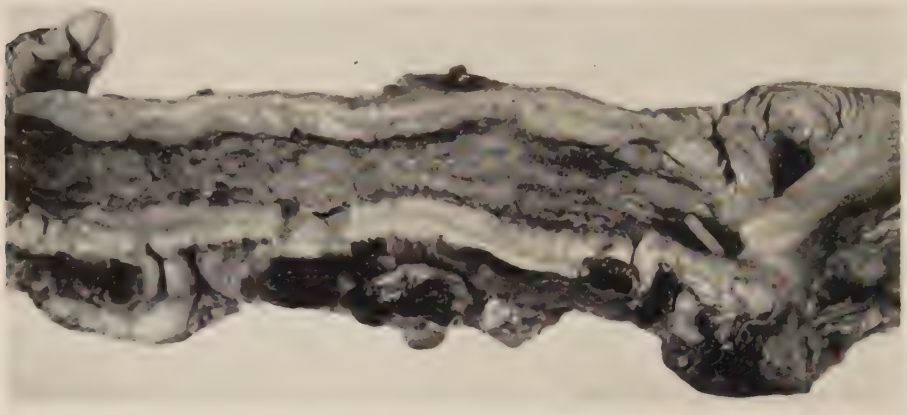

FIG. 7.—Case 1. (×½.) Specimen of chronic ileitis showing ulcerations of mucosa and greatly thickened and edematous wall. Match stick indicates sinus from cecum persisting after appendectomy.

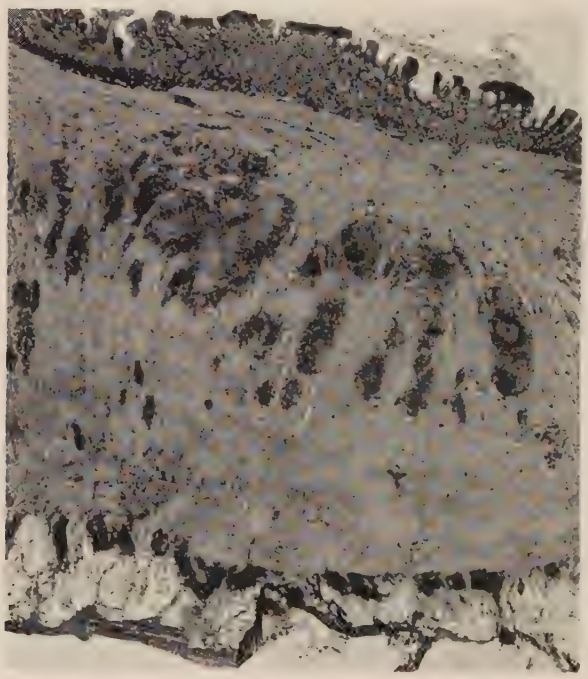

FIG. 8.—Case 1. (×7.) Serosa thickened by areolar and fibrous tissue. Muscles greatly thickened by edema and inner circular layer heavily infiltrated with fibroblasts and clusters of lymphocytes. Lacteals and submucosal lymphatics thrombosed and engorged with large pale mononuclear cells.

large mononuclear cells without identifying their location in lymphatic vessels. The serosa was thickened, fibrinous, and infiltrated with round cells.

Case 3.—A man (M. P.), aged 40, suffered a severe blow on the abdomen from

the steering wheel of his automobile. Two weeks later occasional severe abdominal cramps developed, frequently associated with vomiting and diarrhea. Two months later the cramps were occurring day and night and, on admission, visible peristaltic waves were seen, and a mass in the left lower quadrant was felt. On operation for intestinal obstruction, one of us (F. L. R.) found an area of ileum, which had apparently been traumatized at the time of the accident, bound down by fibrinous adhesions and kinked. Proximally the small bowel was hypertrophied for a short distance, so that some 12 inches were resected. The mesentery was very short, boggy and thick, and at one point in its root a small mass of dark colored material, either fecal material or old unabsorbed blood pigment, was found.

Pathologic study of the resected ileum showed the mucosa to be intact (Fig. 9 p) with a heavy lymphocyte infiltration at its base extending into the submucosa which was greatly thickened and endematous, with fibrin deposits, clusters of lymphocytes and

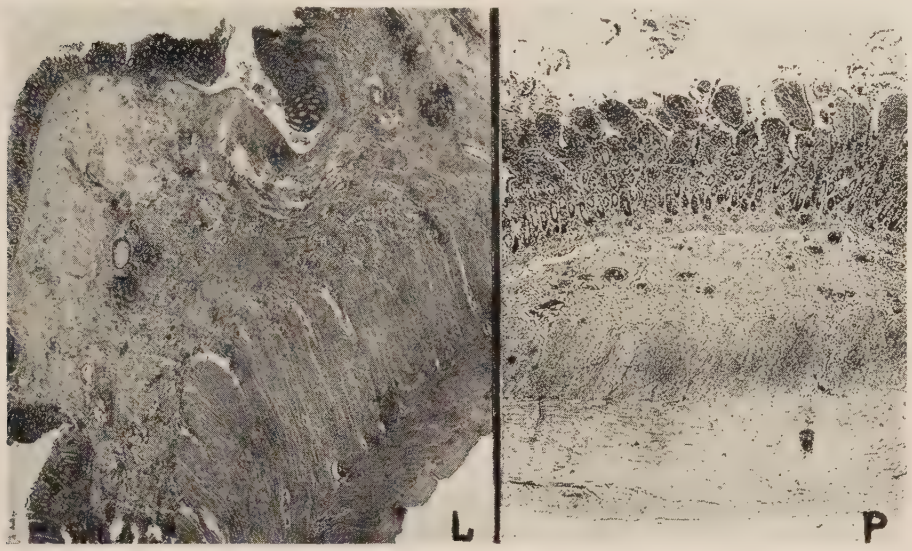

Fig. 9.—Case 2. L (×12). Chronic ileitis. Submucosa edematous and infiltrated with lymphocytes. Muscle layers greatly thickened and distorted with edema. Moderate lymphocytic infiltration. Serosa fibrinous and infiltrated with round cells. Lacteals engorged and many submucosal lymphatics thrombosed. Case 3. P (×20). Chronic ileitis. Submucosa greatly thickened and edematous, with clusters of lymphocytes and with engorged and thrombosed lymphatics. Muscles thickened and edematous. Lacteals engorged, with many thrombosed.

engorged and thrombosed lymphatic vessels. The muscles were quite edematous and thickened with infiltration by round cells and lymphoid tissue. The engorged lacteals between the muscle layers were prominent and many were thrombosed. The serosa was thickened by a heavy infiltrated fibrinous coat.

Discussion.—From a careful gross and microscopic study of the specimens of regional enteritis and the specimens of chronic intestinal lymphedema produced experimentally, a definite and close similarity was seen. Although the pathologic changes were more pronounced in the human cases, yet, in both the clinical material and in the material experimentally produced by chronic lymphatic obstruction, the resemblance was marked in the pathologic alterations seen in the submucosal and muscular layers and in their lymphatic

vessels. No such resemblance had been obtained by Bell[8] when he interfered with the blood supply of the intestinal tract in animals.

In our experiments the sclerosing material was injected only into lymphatics, and we feel that the pathologic changes resulting from such injections were due entirely to the lymphatic sclerosis and obstruction, since we found no evidence of blood vascular thrombosis in the microscopic sections.

The appearance of the gross specimens of regional enteritis and of experimental intestinal lymphedema, although varying in the degree of involvement, show the same thickened, edematous walls and microscopically greatly thickened muscular and submucosal layers which are edematous and have engorged and thrombosed lymphatics and lacteals.

The more extensive stenosis and mucosal ulceration seen in the human specimens might be attributed to the persistence of a chronic low grade bacterial infection. Our greatest thickening of the intestinal wall (Fig. 3) was secured when bacteria had been given intravenously shortly before the lymphatic injection.

It was surprising to find that months after such a lymphatic obstruction, although the animals appeared healthy, the pathologic alterations should persist and show no evidence of subsidence. Some of the animals, however, were sacrificed because of emaciation, or signs of obstruction due to the thickening of the injected bowel, or to adhesions kinking the bowel.

The surgical treatment of regional enteritis by resection of the involved portion of bowel is rational, since in the animal with chronic lymphedema, but without evidence of chronic infection, the pathologic alterations are apparently permanent.

Summary.—Chronic lymphedema was experimentally produced in various regions of the gastro-intestinal tract.

This present report deals only with chronic lymphedema of the ileum and colon which was secured by injections into the mesenteric and subserosal lymphatic vessels of irritating and sclerosing materials.

Such lymphatic injections produced sclerosis and thrombosis of the lymphatics, which led to a chronic lymphedema.

Chronic intestinal lymphedema was secured by one injection, or by repeated injections.

Thickening and edema of the intestinal wall occurred and were most marked in the submucosal and muscular layers where the thrombosed lymphatics and lacteals were engorged with large pale mononuclear cells.

The injection of bacteria intravenously in conjunction with lymphatic injections produced the greatest thickening of the intestinal wall.

Intestinal lymphedema was found to persist for ten months without any evidence of subsidence and the pathologic changes appeared to be permanent.

In regional cicatrizing enteritis the thickening and edema were most marked in the submucosal and muscular layers, where engorged and thrombosed lymphatics and lacteals were found.

We believe that there is a close resemblance in the pathologic changes

seen in chronic regional enteritis and in experimental intestinal lymphedema.

The more extensive stenosis and mucosal ulceration in regional enteritis might be attributed to the persistence of a chronic low grade bacterial infection.

The two dominant features of regional cicatrizing enteritis seemed to be a low grade chronic infection with a concomitant chronic lymphedema.

The surgical treatment of chronic regional enteritis by resection has a rational basis, since experimental chronic lymphedema of the ileum and colon apparently is a permanent pathologic alteration.

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DISCUSSION.—DR. JOHN HOMANS (Boston, Mass.).—The experimental observations of Reichert and Mathes are particularly interesting to me because they have made use of what might be called physiologic methods in studying an important system of the body, namely, the lymphatic system,

particularly in its relation to a rather mysterious and interesting disease. As Doctor Reichert has stated, investigators have studied the blood-vascular system in similar ways for many years, but have neglected the lymphatics which, though we hear very little about them, are, after all, intimately concerned with drainage and detoxification of the tissue fluids in many parts of the body. Since a damming up of these fluids is very likely to lead to fibrosis, is it not reasonable that a peculiar state of swelling sclerosis and ulceration should be studied from the standpoint of lymphatic occlusion? Just as Cecil Drinker's methods of cannulating and destroying lymphatics have thrown new light on elephantiasis, or these experiments of Reichert's have suggested that regional enteritis, or whatever one chooses to term it, is dependent, in form, at least, upon lymphatic obstruction. Doctor Reichert has followed up his training with the late Doctor Halsted and with Doctor Sabin in a most ingenious way. I doubt whether others less skillful can produce similar results.

He has certainly shown how very readily edema and sclerosis of the bowel can be brought about by filling the lymph vessels of a region with a sclerosing medium. It is unfortunate that animals, particularly dogs, are so resistant to pyogenic bacteria. This resistance may retard the further verification of an hypothesis which Doctor Reichert has so modestly put forward, though of course he has shown how the presence of bacteria may fortify his results. In any case, he has done mechanically what nature probably does through the intestinal bacteria. Pathologically, regional enteritis is not an entity. Its variations may be due to a changeable exciting factor, for which Doctors Reichert and Mathes have supplied the anatomic background.

Dr. Alfred Blalock (Nashville, Tenn.).—I should like to speak very briefly of some experiments which I think corroborate the findings that have just been reported by Doctor Reichert. These studies were performed by Dr. R. S. Cunningham, Dr. C. S. Robinson and myself.

We were attempting to produce traumatic asphyxia, and we ligated the superior vena cava of a dog. Much to our surprise, nothing happened immediately. About ten days later the dog became short of breath. He died the following day, and at autopsy 900 cc. of milky fluid were found in the pleural and pericardial cavities. We thought possibly it was an accident and repeated the experiment on approximately 50 animals, and chylothorax resulted in more than one-half of them. We then wondered if we could produce lymphatic occlusion without venous obstruction. Complete lymphatic obstruction was produced in three of more than 50 animals studied, by a variety of procedures which included ligation of the ducts on the right and left sides of the neck, ligation of the thoracic duct in the chest, destruction of the cisterna chyli in the flank, and in some cases direct attacks upon the mesenteric lymphatics.

As was stated, we produced complete occlusion in three dogs. The evidence was this: The dogs lost weight. The eosinophils and the lymphocytes disappeared from the blood stream. Chemical studies revealed an abnormality in the absorption of fat. The dogs died, and at autopsy striking changes were observed.

In part of the intestinal tract from one of these three animals, the entire wall was perfectly white. Enormous lymph vessels were observed everywhere. Although the peritoneal cavity was not entered in this experiment, this condition resulted.

In a section of the omentum, large lymph vessels were seen. These were much more marked at the time of autopsy. In addition, there was a pooling of lymph in many of the tissues; for example, in the pancreas. The same

was true of the entire intestinal tract, and even occurred beneath the epicardium of the heart.

A section from a second experiment showed the lymphatics filled with coagulated material. Again the peritoneal cavity was not entered, showing that the same condition which Doctor Reichert can produce by a direct attack can be caused by operating elsewhere.

In the experiments in which complete obstruction was not produced, collateral lymph channels were demonstrated at autopsy. Most of these communications were with the inferior vena cava near the orifices of the renal veins.

Dr. Frederick L. Reichert (San Francisco, Cal.) closing.—Answering Doctor Homans' question about the acute cases of enteritis, I feel that certainly a number of them must recover from the condition without any symptoms, just as that last animal did, living for ten months without any symptoms whatever. Many of our cases probably do recover, but there are others, just as in this series of dogs, in which the thickening continued, some of them having obstruction and others developing adhesions and kinking and shortening of the mesentery. In none of our animals did we find any free peritoneal fluid.

Doctor Blalock's work is excellent. He too has produced thrombosed lymphatics and he has applied this method to the lymphatic system of the entire body. I will expect to hear a great deal more from him.

THE REDUCTION OF THE INCREASING MORTALITY AND MORBIDITY IN ACUTE APPENDICITIS

Hugh McKenna, M.D.

Chicago, Ill.

Dr. John B. Murphy was responsible for establishing a plan for the early diagnosis and immediate surgical management of this disease. Unfortunately, the Murphy plan has not been carried out by the general profession according to his original program. It may be of some interest to know that a few days before the death of Doctor Murphy, during a discourse on bone and joint surgery, he switched abruptly to the consideration of the present status of acute appendicitis, stating that: "Twenty years ago, I was of the opinion that we had taught the profession the vital importance of early diagnosis in acute appendicitis and the immediate treatment by surgery, but in this we have failed and I propose when I get back to work in September, to teach in my clinic, and write upon, the imperative necessity of early recognition of this disease, followed immediately by surgical management." The only charitable thing that may be said respecting delay in the surgical management of this disease is based upon the supposition that in the early period of the evolution of the treatment of acute appendicitis, there were areas where competent surgical services could not be secured. Whatever interpretation may be placed upon delay in action, a review of the literature shows that disastrous results have followed procrastination, in diagnosis and treatment, of this disease.

Until the medical profession makes an attack on the problem of acute appendicitis from a different point of view than has generally been done, no marked improvement will result. The work done by Bower and his associates[4, 5] gives conclusive evidence of this fact. Most of the teaching and writing is occupied with the management and treatment of the pathology of a disease that should never occur. It seems just as illogical to attempt to improve the mortality rate in acute appendicitis by teaching how to handle extensive pathology, abscess formation and peritonitis, as it would be to go back to the period, before 1883, when Klebs discovered the bacillus of diphtheria, and to attempt any marked improvement in the treatment of diphtheria by refined methods of handling the pathology resulting from the disease rather than by preventing the stages of severe pathology.

The foregoing is not a parallel simile in disease types but it suffices for the purpose of calling the attention of the profession to the imperative necessity of attacking the problem of acute appendicitis in its inception and at a period when minimum pathologic changes have taken place. This program calls for much of the time and energy, utilized in teaching the methods used in treating the pathology caused in the late period of the disease, to be expended in an organized movement to make the lay people appendicitis con-

scious. Poor surgery, as demonstrated by the "occasional operator," much as that type of surgery may be deplored, is not responsible for the distressing rapidly increasing mortality rate in acute appendicitis.

When one reviews the voluminous literature on acute appendicitis he is impressed with the seriousness of this situation. However, when the mortality statistics taken from the U. S. Department of Commerce, Bureau of the Census, 1934, are reviewed the situation is not only serious but almost unbelievable (Table I).

TABLE I

THE DEATH RATE PER 100,000 ESTIMATED POPULATION IN THE REGISTRATION STATES OF 1900–1930

CAUSE OF DEATH — APPENDICITIS

1900	1910	1919	1920	1921	1922	1923	1924	1925	1926	1927	1928	1929	1930
8.8	11.1	11.7	13.2	14.2	13.7	14.4	14.9	15.1	14.8	15.0	15.2	15.9	15.8

Statistics are usually uninteresting and writers usually try to avoid them, especially in papers. However, I shall attempt to give you a better idea of just what these figures mean: A mortality of 15.8 individuals in each 100,000 means 158 in each one million, and since in the United States we have a population of 122 millions, this means a mortality in this country of approximately 20,000 individuals dying each year from this disease and its complications. To more clearly visualize what this loss of life means, the dead would be approximately equal to twice the number of men in the Illinois National Guard. It is appalling to consider this tragic condition in a disease that should, under proper control, be brought to a small fraction of 1 per cent.

The foregoing narration does not take into account the morbidity in connection with acute appendicitis. What are some of the factors that lead to the increasing mortality in acute appendicitis in the United States? How does this compare with the death rate in other countries?

Again, quoting from Bower's statistics the incidence of appendicitis in the United States is:

TABLE II

59.3 per cent higher than the City of Mexico
70.0 per cent higher than Germany
70.0 per cent higher than Scotland
98.7 per cent higher than New Zealand
109.5 per cent higher than England and Wales
131.8 per cent higher than Irish Free State
313.5 per cent higher than Italy

Doctor Hoffman, consulting statistician of the Prudential Life Insurance Company, says: "During 1932 in 177 cities with a population of 43,021,704 there were 7,136 deaths, a mortality rate of 16.6 per 100,000." Still further he says, "In 1932 in ten of the largest cities of Pennsylvania, exclusive of Philadelphia, with a population of 1,474,567 there were 301 deaths, a mortality rate of 20.5 per 100,000. In 1931 these same cities with a population of

1,460,063 had 255 deaths, a mortality of 17.4 per 100,000. Philadelphia in 1931 with a population of 1,966,351 had 274 deaths, a mortality of 13.9 per 100,000; and in 1932 with a population of 1,978,663 there were 223 deaths, a mortality of 11.3 per 100,000." Bower[4] from whose paper I quote these statistics, is of the opinion that the lowered mortality rate in Philadelphia is due to the campaign carried on in that city.

Etiology.—Geographic.—With Social and Individual Characteristics of Living.—If the mortality in appendicitis is to be reduced, consideration must be given to the comparative incidence of the disease in this country, as compared with other countries; some deductions may be made in the geographic factor by comparing the relative frequency of appendicitis in the white and black races. Boland[3] reviewing 4,270 cases in Atlanta found that the disease was six times as common in the white as in the colored race. Commenting upon the relative infrequency of appendicitis among colored people, he draws attention to the report of McCarrison, in India, where the disease did not occur among several thousand patients who lived upon "natural foods" free from preservatives. Most Negroes in the south live on cornbread, peas, cabbage and turnip green juice (pot likker). Thirty years ago appendicitis was almost a novelty among these people, but as they gave up simple food the disease has increased, according to Boland. The factors pointed out in this review may explain the increased incidence of the disease in the United States as compared with the countries referred to in Table II.

Age Incidence.—Appendicitis is primarily a disease of the young, the greatest incidence between the ages of 11 and 20, although it may occur at any age. Hudson[19] shows that "in Massachusetts appendicitis was recorded as the cause of death in 1,795 children from 1900 to 1930. In 1900 the diagnosis was recorded 25 times, and in 1930 107 times. This represents an increase of 428 per cent in a period in which the population increased only 41 per cent." According to his report appendicitis held eighth place as a mortality factor in children.

Habits of life and foods leading to constipation.

The hemogeneous factor in connection with acute tonsillitis, and upper respiratory tract infections, in my opinion, has not received sufficient consideration. Other foci of infection should not be overlooked.

Symptoms and Diagnosis.—Wilkie has directed attention to a point in differential diagnosis between the acute inflammatory and the obstructive form. In brief, he describes the obstructive form as coming on with severe cramps, usually violent in character, followed by nausea or vomiting, with usually repeated colicky attacks. The inflammatory form he describes as coming on with the symptoms of malaise, without sharp pain. He considers the obstructive type as the more serious condition.

In a fairly extensive experience in acute appendicitis during the war it was interesting to note that with medical officers drawn from many parts of the country, few of them were trained in the importance of securing a history of symptoms in sequential order. Acute appendicitis begins *with pain*

and as a rule up to 18 hours the pain is referred to the pit of the stomach, finally localizing in the region of the appendix which in the large percentage of cases is in the lower right quadrant of the abdomen. Diagnosticians should not overlook the fact that the appendix may be in other regions of the abdomen: namely, (*a*) the epigastric region, (*b*) anywhere in the left abdominal region, (*c*) and not infrequently in a position immediately in front of the right kidney. In this latter position it not infrequently leads to a diagnosis and formation of a perinephritic abscess. (*d*) The appendix may be found in any of the normal openings of the abdominal wall, such as the inguinal or any other hernial opening.

In the second attack nausea or vomiting or both may be absent. I have already published what I believe to be the reason for the absence of these symptoms, namely, the fixing of the cecum by the inflammatory reaction consequent to the first attack.

Leukocytosis is an important factor if properly interpreted. Keep in mind that the white count may be positive or only relative. As I previously demonstrated in a report, with Morris, on 234 soldiers operated upon at Camp Pike Base Hospital, the white count was only relatively high. We concluded that since these soldiers had received typhoid and paratyphoid vaccine, previous to the attack of appendicitis the blood would show a leukopenia. We ran a control upon 100 soldiers in normal health, who had previously received inoculations of typhoid and paratyphoid vaccine, and demonstrated that a leukopenia existed. It was evident therefore, that when these individuals suffered an attack of acute appendicitis an increase to 8,000 white cells would represent a relative leukocytosis. With an initial white count of more than 22,000, careful search should be made to find a possible cause outside the appendix, particularly the possibility of a pneumonic process.

Temperature may be increased at the beginning but not necessarily so, and absence of temperature should not mislead one in making the diagnosis. Later when the inflammatory process is well established or is spreading, the temperature curve is of importance. The pulse rate is not necessarily of diagnostic importance at the onset.

I have not found rigidity of the abdominal muscles of importance in the very early hours of the attack during the most favorable period to make the diagnosis. One should never fail to make a rectal examination, especially in children. I have always considered this of importance in children in making a differential diagnosis between appendicitis and possible pneumonia.

Morbidity and Pathology.—Morbidity is so dependent upon pathology that these subjects are treated conjointly. In Wilkie's investigations, unquestionably, a point was made in differential diagnosis based upon the pathology in the appendix. The obstructive type, made possible in many instances because of a previous inflammatory condition, produces an attack through the formation of a fecalith distal to a constriction near the base of the appendix, which cannot pass and in this position rapidly cuts off the blood supply. A similar pathologic change may be brought about by a con-

stricting band at or near the base of the appendix. I trust the teaching of Wilkie may not lead to any delay in the surgical management of the acute inflammatory type of appendicitis. In the first place, the differential diagnosis may not always be made and, at any rate, the inflammatory attack makes the pathologic change upon which the obstructive type is formed.

Treatment will be considered under the following heads:

(1) Education of the people of the possible seriousness of pain in the abdomen, especially in the young.

(2) A campaign in the medical profession on the necessity of immediate hospitalization of patients suffering with pain in the abdomen where a tentative diagnosis of appendicitis is made.

(3) The type of treatment to be instituted.

Thanks to the efforts of Bower, the mortality of acute appendicitis has been greatly reduced due to the campaign carried out in Philadelphia. In brief, the plan consisted in making the public appendicitis conscious by a publicity campaign. Through the Philadelphia Association of Retail Druggists the following placard was placed in most of the drug stores of that city:

APPENDICITIS

SEVERE PAINS IN THE ABDOMEN ARE OFTEN DANGEROUS. DO NOT TAKE PURGATIVES. CALL A PHYSICIAN. DEATHS FROM APPENDICITIS ARE INCREASING ANNUALLY.

In addition, these placards were sent to family physicians. Recognizing the greatest incidence of the disease between the ages of 11 and 20, talks were given in schools and sticker placards sent them directly to be posted in their school books. Under the head of warning they issued the following placard:

WARNING

IN THE PRESENCE OF ABDOMINAL PAIN:
NEVER GIVE A LAXATIVE.
GIVE NOTHING BY MOUTH.
APPLY ICE CAP OR WATER BOTTLE.
CALL YOUR FAMILY PHYSICIAN.
ABDOMINAL PAIN WHICH LASTS MORE THAN SIX HOURS IS USUALLY SERIOUS.

The Philadelphia plan was published by the Philadelphia County Medical Society and endorsed by the Department of Health. The plan was given much publicity and, by lectures to the laity, an attempt was made to show the relative seriousness of the clean and peritonitis case.

Recommendations.—(1) That a plan similar to the Philadelphia campaign be instituted by every county medical society, using every legitimate avenue

open to organized medicine to reach the public respecting the seriousness in delay in recognizing and treating acute appendicitis.

(2) Give more attention to this subject in undergraduate teaching. Arrange to have a paper on this subject before every branch society annually. Invite all hospital staffs to check the annual death rate from acute appendicitis with the hope that the community, both lay and medical, may become appendicitis conscious.

(3) The treatment is surgical in the large percentage of cases. Dixon, of the Mayo Clinic, has expressed the belief that "the increase of deaths from appendicitis is due to three factors, two of which concern the medical profession, while the third concerns the unfortunate widespread use of cathartics by the patient with appendicitis." Dixon believes that more patients with appendicitis have been operated upon by the "occasional operator" during the past ten or 12 years; he also suggests that the present generation of younger surgeons has not taken seriously the wide experience of the older surgeons in this field. The tendency of some physicians to regard appendectomy as a minor surgical undertaking should be universally condemned. There is no operation in the whole realm of major surgery which may demand greater exercise of surgical skill and judgment.

When I say the treatment is surgical I do not wish to say that in the pus or local peritonitis cases that the same surgical procedure should be followed that obtains in the clean cases. I am firmly of the opinion that if a reduction in the mortality is made in the late cases, where rupture has taken place, it must come in determining first the cases that should be operated upon, and secondly, the type of operation to be instituted.

There is no doubt that many patients diagnosed as progressing peritonitis or general peritonitis have only localized peritonitis, and in many cases this cannot be determined without operation. In these borderline cases, to make a small opening under local anesthesia and introduce a drain should not add to the mortality. On the other hand, even through a small opening, without introducing anything into the peritoneal cavity, a localized peritonitis may many times be diagnosed and the pus drained, and a general peritonitis prevented.

In the cases with a localized abscess I have always attempted the plan of entering the pus cavity without entering the free peritoneal cavity. This can be done only in a small percentage of the pus cases. In all other localized pus cases the surgeon can wall off the peritoneal cavity in a thorough and systematic manner, so that the pus may be evacuated and gently sponged out with gauze sponges soaked in one-half of one per cent lysol solution, drains inserted to the bottom of the cavity, and the omentum carefully drawn around the drain and the wound closed. This procedure can usually be carried out without the introduction of anything into the free peritoneal cavity by the moist sponges put in position before the pus cavity is opened. I follow the rule of taking the appendix out in these pus cases when the line of cleavage through which the pus cavity is entered leads to the appendix. In

this procedure the appendix may be removed without soiling the free peritoneal cavity more than in the drainage operation. In the large percentage of cases the ruptured appendix should be removed, which in my opinion if taken out by the plan outlined does not increase the mortality, and decreases the morbidity.

An entire paper might be advantageously given to a detailed discussion of many special methods of treatment, such as high enterostomy, introduction of a catheter into the cecum, Fowler's position, with a "rectal drip" of normal saline solution by means of a properly arranged douche tip.

At Camp Pike, during the war, I was able to get the Division Surgeon to issue an order that all soldiers suffering with abdominal pain, that persisted, be sent to the hospital. In approximately 450 operations upon soldiers, the large percentage of which were for an acute appendicitis, the mortality rate was markedly reduced, and the fatalities that occurred were due to either an inability to gain consent to perform the operation, or to some complication.

The morbidity and mortality in acute appendicitis will be reduced when the disease is brought to operation within the first few hours following the initial attack. In the doubtful cases call a consultation early in the disease, and if the diagnosis cannot be made, err on the side of surgical intervention.

Sufficient attention has not been directed to appendicitis developing in unusual locations in the abdomen, and particularly that which, because of location, becomes the etiologic factor in perinephritic abscess.

ABBREVIATED, ILLUSTRATIVE CASE REPORTS

Case 1.—Mr. R. entered St. Joseph's Hospital giving a history of having been operated upon for a right perinephritic abscess one year previously. A permanent fistula, discharging pus, followed this operation. A thorough examination of the genito-urinary tract revealed no connection with the fistula. A barium enema demonstrated that the fistula was connected with the right colon. An abdominal operation disclosed an old ruptured retrocecal appendix lying on the anterior surface of the right kidney. The remnant of the appendix was removed and the patient made an uneventful recovery, almost 15 months following the original attack of appendicitis. Consider if you will the economic waste caused by a mistaken diagnosis.

Case 2.—A young male patient was brought into the receiving ward of the Cook County Hospital. The receiving intern, a student, announced in the clinic that if my teaching was correct this patient had appendicitis, although his physical symptoms were those of a perinephritic abscess. Basing the diagnosis principally upon the history the patient was operated upon immediately for acute appendicitis. When the cecum and base of the appendix were exposed these structures appeared normal. However, as the appendix was traced back in its retrocolic position and freed from adhesions, the tip passed for a distance of one and one-half inches through and behind the retroperitoneum. Cutting through this structure the distal end of the appendix was found with a necrotic wall and abscess formation, and containing a fecolith, the mass lying just anterior to the right kidney. Had this young man not been operated upon immediately a perinephritic abscess would have formed and a subsequent operation in the lumbar region would have corroborated the wrong diagnosis just as in the first case cited.

Case 3.—A boy, aged 17, the son of a physician and the grandson of a noted medical teacher, was admitted to the hospital after an illness of about five weeks. A number of the leading specialists of the medical school in which the grandfather had taught had seen this patient. There was a very classical history of appendicitis recorded, but on

Table III

CASES OF APPENDICITIS — ST. JOSEPH'S HOSPITAL

1922–1935

Diagnosis	1922	1923	1924	1925	1926	1927	1928	1929	1930	1931	1932	1933	1934	1935	Total
Appendicitis, acute	35	32	36	42	58	48	50	76	79	78	62	49	77	55	777
Appendicitis, gangrenous	22	24	27	33	39	35	31	25	22	30	34	18	33	45	418
Appendicitis, chronic	82	73	67	80	116	104	102	93	89	97	73	56	63	56	1,151
Appendicitis, subacute	16	22	23	18	17	25	29	35	23	26	17	16	31	32	330
Appendicitis with peritonitis	6	7	7	4	6	7	4	3	4	3	1	3	2	5	62
Appendicitis. Incidental appendectomy	35	39	22	30	36	40	37	37	54	40	18	24	19	18	449
Total	196	197	182	207	272	259	253	269	271	274	205	166	225	211	3,187

Total deaths, 78; mortality, 2.48 per cent.

APPENDICITIS — DEATHS FOLLOWING OPERATIONS — 1922 TO 1935

Appendicitis, acute suppurative	9
Appendicitis, gangrenous, ruptured	36
Appendicitis, gangrenous, with peritonitis	15
Appendicitis, with abscess	3
Appendicitis, chronic	12
Appendicitis, subacute	2
Appendix, incidental—cyst of ovary	1
Total	78

CAUSES OF DEATHS

Dilatation of heart, acute	1
Embolism, pulmonary	4
Myocardial failure, acute and shock	1
Myocarditis and nephritic, chronic	1
Myocarditis, chronic	1
Obstruction, intestinal, acute	1
Paralysis, cardiac, following embolism	1
Paralytic ileus	17
Peritonitis, general	34
Pneumonia, broncho	1
Pneumonia, lobar	4
Psychosis, acute exhaustion, and hypostatic pneumonia	1
Parotitis, bilateral, septic with meningitis	1
Septicemia	2
Septicemia and myocarditis with multiple emboli	1
Shock, surgical	5
Toxemia and pneumonia	1
Total deaths	78

Mortality, 2.48 per cent; total cases, 3,187.

physical examination the pain was very low down in the pelvis and on the left side. As a result the diagnosis was not made. On the morning the patient entered St. Joseph's Hospital in the fifth week of his illness, in order to complete the physical examination a roentgenologic examination was made of the chest. This was negative. In a few hours following admission the patient had a sudden, severe, explosive coughing attack, with rapid respiration, increased pulse rate, elevation of temperature, and in short the signs of marked chest pathology, which the physical findings and the subsequent roentgentogram corroborated. The patient developed a pneumonia followed by empyema and a long drawn out convalescence, being confined to the hospital approximately three months. At the time of discharge from the hospital the father was instructed in the event of abdominal pain to immediately bring the patient to the hospital for surgical treatment.

Approximately six weeks following his discharge, the patient had an attack of abdominal pain. He was hurried to the hospital and an emergency operation was performed. Through a lower left rectus incision a necrotic appendix was removed from a bed of old adhesions on the left side of the pelvis in the rectovesical cul-de-sac. Recovery was uneventful. Consider what this patient went through—his suffering, serious condition, expense, and loss of time—all because a correct diagnosis was not made in the first few hours of the disease and the inflamed appendix removed.

The recitation of these three cases illustrates the pathology which may result from acute appendicitis, where the patient, although seen early by a physician, was not correctly diagnosed, and was not afforded the relief which immediate operation would have effected.

Doctor Murphy, who continued to teach the imperative necessity of early diagnosis and immediate operation in the treatment of acute appendicitis, made the following comment April 7, 1915: "Just recently a critic took a Chicago surgeon to task in the columns of a medical journal because the latter had published a colored picture of a gangrenous appendix in connection with a practical talk on the proper treatment. The critic intimated that appendicitis was ancient history. It is ancient history; but does that statement mean that all practitioners are masters of the subject, or that the disease is efficiently handled at the present time?

"In looking up recently for the Year-Book of Surgery the hospital statistics on the results of operation for appendicitis, what mortality rate do you suppose I found—the hospital mortality rate? Someone guess! I do not mean the mortality for one, two, or three splendidly equipped hospitals, with staffs composed of the leaders of the surgical profession; I am speaking of the combined statistics of a number of hospitals in the United States. That average hospital mortality rate is just a little over 10 per cent!

"Is it time to stop talking about appendicitis? No! It is just the time to begin talking about appendicitis, and talking more seriously and emphatically about it. When you know that in our best hospitals better than 98 per cent of all the acute appendicitis cases, including those with abscess and peritonitis, are saved, and when you know that scarcely one out of a hundred of the cases of acute appendicitis operated upon during the first 24 hours of the attack is lost, I think what the results must be in the other hospitals to make the general average so appalling. There is no palliative excuse for a mortality of 10 per cent in appendicitis. That rate is simply shocking. Even cases of

appendicitis with perforation into the free peritoneal cavity have now but a very slight mortality when operated upon in time by experienced hands; and still the appalling combined rate is 10 per cent, including the chronic and interval cases—which should show practically no mortality at all—as well as the acute cases. These patients did not die because of the operation—they died in spite of it. They died, not so much because of any fault in technic as, because of the fact that they did not reach the hospital in time for a successful operation. Procrastination was the cause of death—the almost criminal cause. The initial symptoms are clean cut and almost unmistakable. The mode of onset of an attack of appendicitis is no clue to its probable course or complications. We can never tell in a given case what the next day may bring. Therefore, operate today. By operation we take the course of the disease into our own hands. By not operating we leave the case in the hands of a blind and often terribly cruel fate."

After reviewing hospital statistics showing the mortality statistics in acute appendicitis following various methods of classification, it was interesting to review the report from St. Joseph's Hospital, Chicago. In this hospital for many years the watchword has been "early diagnosis in acute appendicitis and treat immediately by surgery." The following report on 3,187 cases includes all operations where the appendix was removed, 1922 to 1935 inclusive, acute, chronic and incidental. These operations were performed by staff and nonstaff members.

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RESECTION OF THE RECTUM AND RECTOSIGMOID BY SINGLE OR GRADED PROCEDURES

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The routine application of stereotyped technical procedures to any pathologic process, especially if that process be a malignant one, should be and is discountenanced by all experienced surgeons. Nowhere is this more forcibly emphasized than in the selection of surgical measures for the radical extirpation of cancers in the lower gastro-intestinal tract.

That no one type of offensive meets the requirements of all cases is axiomatic, but it is, or should be, equally true that the majority of these cases should be operated upon by as radical surgical procedures as are compatible with a reasonable hospital mortality. To increase the scope of operability is as imperative a duty as to reduce the hospital casualty list, and to this end several surgical operations either in one or more stages must be included in the surgeon's campaign plans.

Statistical studies by Miles, Jones, and others have proved incontrovertibly that the application of the same principles of surgery to cases of cancer of the rectum as to cases of cancer of the breast, lip, *etc.*, yields immensely higher percentages of five year cures than the less extensive procedures. That the radical operative maneuvers may not be applied to all cases of cancer of the rectum which are deemed operable and that they cannot be applied always in the ideal operative method, is, I think, hardly debatable. Consequently, while one deprecates multiple surgical procedures, particularly those which demand reopening of the abdomen at a second stage when one operation will accomplish the same result, there is small question that a field does exist not only for the graded radical extirpative procedures, but also for the less formidable operation of colostomy and posterior resection.

The choice of offensive against cancer of the rectum and rectosigmoid, viewed from the operative standpoint as well as from the standpoint of end-results, is, in my judgment, the one stage combined abdominoperineal resection of the rectum following the technic of Mr. Ernest Miles. That the mortality figures of this type of operation cannot be reduced beyond a range of 5 to 10 per cent even in the most experienced hands, without a too noticeable reduction in operability rate, will, I think, be admitted. Its mortality rate has been the one objection to the operation, but I am convinced that this objection is no longer defensible because of the low mortality statistics of experienced surgeons which my own recent experience in a series of 22 cases operated upon without mortality supports.

More familiarity with technical details and more meticulous preoperative and postoperative care have allowed me to utilize this one stage maneuver in a higher percentage of cases than hitherto. That this ideal may not be

carried out in many cases where the rectal tumor is removable, yet hazardously so because of complicating coexisting debilitating diseases and the patient's inability to stand formidable operative procedures, is quite evident.

For the less sturdy risks—and this is a fairly numerous group of cases—I feel that a two stage maneuver of the type advocated for many years by Daniel F. Jones, or the technic of a graded radical combined abdominoperineal resection, which in reality is a modification of Miles' technic into two stages, as described by me[2] in 1929, has a definite field of usefulness. In this maneuver the bowel is divided, turned in, the distal segment dropped back, and a single barreled colostomy made in the left flank. Lahey one year later modified my operation by bringing out the lower end of the bowel and using it to irrigate through. I have not utilized this procedure because I have felt that if obstruction was present one divided the bowel at a greatly increased hazard and if obstruction was not present in a marked degree, one could irrigate with a two way tube in the rectum just as satisfactorily.

This two stage operation, I am confident, has allowed me to perform a radical operation in many cases where the growth was so large and inflammatory or where the patient's condition was so debilitated and undermined that a single stage operation would have been attended by a prohibitive mortality whereas the only other choice was a colostomy and subsequent posterior resection.

That the graded operation can be done in this group of cases to possibly extend the operability with the same or slightly lower operative mortality, is my belief. Nevertheless, I must confess that within the past two years my own experience with the one stage operation has led me to substitute it in an increasing number of cases, to the virtual abandonment of other types except under the circumstances mentioned above.

The second variety of graded maneuver which still holds a place in surgery for cancer of the rectum is the operation of Mummery—that is, colostomy and subsequent posterior resection. That this operation does not remove the lymphatic nodes of the sigmoid mesentery and that it is far from being a radical type of procedure, one cannot deny, yet it can incontrovertibly be carried out in a group of cases which are such grave operative risks that the radical procedures are not to be considered, and with five year cures of 38 per cent as I[3] showed in reviewing 300 cases.

Because of slow metastases from rectal cancer, those occurring in the ampulla unquestionably can many times be operated upon successfully by this latter method, but there is small question that a statistical study of the end-results will show a much lower percentage of five year cures if this type of operation is routinely employed, especially in the rectosigmoid cases.

The indications for a two stage operation are, I think, defined by first, the patient's general inability to withstand a formidable surgical procedure; second, local complications such as an unusually large tumor with little mobility, or fixation to adjacent viscera; third, cases requiring double resection of large and small bowel or bladder; fourth, anatomic types such as extreme

obesity; fifth, coexisting debilitating diseases such as diabetes and cardio-renal vascular diseases; and sixth, extreme old age.

Given a movable growth in the rectum or at the rectosigmoid in an individual without coexisting complications and an average operable risk from a general standpoint, the operative choice is the one stage combined abdomino-perineal resection. For the above mentioned exceptions the two stage combined and the colostomy and posterior resection offer a distinct chance for palliation or cure to a definite group.

In the past three years in my own experience, six changes in operating upon rectal and rectosigmoidal cancers have been established. These are: first, elimination of intraperitoneal vaccination as a routine step in the preliminary preparatory period; second, the abandonment of spinal anesthesia; third, an extension of the period of preparation to at least seven days; fourth, the routine performance of a presacral neurectomy; fifth, routine blood transfusions postoperatively; and sixth, the adoption of the one stage combined abdominoperineal resection in a higher percentage of cases than hitherto.

Intraperitoneal Vaccine.—In 1928, at my suggestion, Herman did some experimental work in Mann's laboratory with intraperitoneal vaccine of colon bacillus and Streptococci, which prompted us to use it routinely in the preliminary preparation of patients operated upon for organic lesions of the large bowel and rectum. Subsequently, after utilizing this vaccine in a series of 60 cases, Bargen and I reported favorable declines in operative mortality statistics, and a smoother postoperative convalescence.

While it was recognized that this was only one of a series of advantageous steps in the rehabilitation of these individuals with colonic and rectal lesions preoperatively, we felt that it had a large sphere of usefulness, and in a further report on vaccination against peritonitis in surgery of the colon we reported a series of 300 cases in which the some favorable outcome influenced us to credit the vaccine with being a large factor in the mortality decline. In a review of 527 surgical lesions of the large intestine and rectum which I published in 1930,[4] the mortality rate was 12.3 per cent by patient and 8.6 per cent by operation.

In February, 1933, I began a series of operations for organic lesions of the colon and rectum in which the preoperative vaccination was not utilized. Table I shows the diagnoses of 130 cases of organic lesions of the rectum and colon which were operated upon in this series without the use of preliminary intraperitoneal vaccine:

TABLE I

DIAGNOSES IN SURGICAL DISEASES OF THE ILEUM, LARGE INTESTINE AND RECTUM

	Patients	Deaths
Cancer	95	8
Diverticulitis	5	0
Intestinal obstruction	5	0
Ulcerative colitis	4	1

TABLE I—*Continued.*

	Patients	Deaths
Megacolon	4	0
Polyposis	4	0
Fecal fistula	2	0
Tuberculosis	2	1
Inflammatory cecal tumor	2	0
Intussusception	1	0
Miscellaneous conditions	6	1
	130	11

Mortality by patient—8.4 per cent.

In 200 consecutive operations done on these 130 patients there were 11 deaths, a mortality of 5.5 per cent by operation, and 8.4 per cent by patient. The pathologic conditions, operative technic, and operability rate utilized in this series were identical with the former series. Table II shows the types of operation done in this series of cases and indicates that both the diagnoses and operations parallel those published in 1930:

TABLE II

TYPES OF OPERATION ON THE COLON AND RECTUM IN 200 CASES

	Operation	Death
Combined abdominoperineal resection, one and two stages	23	0
Posterior resection and colostomy	40	1
Colostomy alone	13	4
Obstructive resection	16	2
Resection right colon	10	0
Ileocolostomy	15	2
Exteriorization (graded)	33	1
Sympathectomy	4	0
Colectomy	3	1
Cecostomy	13	0
Ileostomy	6	0
Enterostomy	2	0
Closure of or plastic on colostomy	9	0
Abdominal exploration	3	0
Miscellaneous	10	0
	200	11

Mortality by operation—5.5 per cent.

This series of cases has convinced me that the decline of the mortality rate under cooperative management which included intraperitoneal vaccination was due more largely to the other rehabilitory and decompressive steps than to vaccine.

That peritonitis is the most common lethal factor following major operations for organic lesions of the large bowel and rectum is a common observa-

tion in all autopsy statistics, and logically one must concede that any measure which increases the resistance of the peritoneal tissues is desirable. That further research will confirm this opinion is an earnest hope, but the present evidence seems to point largely to the question of decompression, hydration, and general rehabilitory measures as the most potent factors in increasing resistance to intraperitoneal infection.

Anesthesia.—Elimination of spinal anesthesia in my service has not been with any regret. While spinal anesthesia has many advantages to the surgeon and perhaps to the patient as well, the inability to control it and occasional surgical accidents have commonly been the reasons for its abandonment.

The ideal anesthetic remains to be evolved but I have found great comfort in the use of gas-oxygen and ether where the one stage operation is decided upon, and gas-oxygen and ether for the first stage of the two stage operation while transsacral and gas-oxygen have been employed in the second stage of the graded maneuver.

With better preliminary medication and better administration of anesthesia, the margin of safety has been unquestionably increased, yet the disappearance of pulmonary complications remains a hope rather than an achievement.

Presacral Neurectomy.—For the past two years I have routinely performed a presacral neurectomy after either the one or the two stage resection has been completed. There are two reasons for this: first, to influence the atony of the bladder; and second, for relief of pain in the event of pelvic recurrences.

The abdominal part of the abdominoperineal resection is a very extensive dissection and there is small wonder that, in cleaning out the fat and soft tissues both from the lateral pelvic walls and the hollow of the sacrum, even greater injury is not done to the nerve supply of the bladder. It is a common experience that complications in the urinary tract following the bladder atony and necessary catheterization are of quite serious import.

Learmonth's[1] work on the innervation of the urinary bladder and his resection of the presacral nerve in the treatment of cord bladder and certain other types of atonic conditions with benefit, pointed logically to the presacral neurectomy as an advantageous step following rectal resections. He found that normal bladder activities are controlled by three chains of nerves, namely, the sacral autonomic, the thoracolumbar outflow of the sympathetic system, and the somatic centers in the sacral part of the spinal cord. Each of these systems contains afferent and efferent fibers. The nerve is easily accessible as it passes in front of the fifth lumbar vertebra, and following ligation of the inferior mesenteric artery with the bowel pulled forward and the peritoneal flaps mobilized, it is a simple matter to ligate the middle sacral artery and then sweep the tissues in front of the sacrum across over the left common iliac vein and upward toward the inferior mesenteric stump.

I have felt that there was a distinct improvement in the emptying of the bladder and a consequent lessening of urinary complications following its utilization. The most reasonable explanation of the success of neurectomy seems to be that the hypogastric nerves in man carry inhibitory impulses to the bladder which may be sufficient to prevent its complete emptying when these nerves are intact and the pelvic nerves are injured.

The reported results of sympathectomy carried out for the relief of pelvic pain seem to indicate that the same procedure would be useful for pain of recurrent malignancy. There is some controversy among neurologists as to the mechanism by which this is accomplished, the only proved contribution of the autonomic nervous system to pain being in relation to referred pain in the production of which only efferent fibers are utilized.

The fact seems well established that pain impulses are mediated along the hypogastric plexuses, and whether they are transmitted over the autonomic nerves or pathways belonging to the spinal nerves is immaterial if by sectioning the hypogastric plexuses the painful impulses can be interrupted and the intense pain of recurrent malignancy prevented.

Blood Transfusions.—Following resections of the colon or rectum, regardless of the type, it has been our custom within the past two years to give a routine blood transfusion of 500 cc. of citrated blood. The physiology upon which this step is based is not clear, but the impression that it is most desirable is confirmed by the smoother convalescence, the absence of any delayed reaction, and the general improved outlook in the cases in which it has been utilized.

Resections of the colon and rectum are not shocking operations if they are done meticulously with accurate hemostasis and on properly selected cases. These people leave the operating table not in collapse, but with a good pulse and blood pressure, and react promptly from the anesthetic in the majority of instances. That the blood transfusions probably tide them over the period where delayed reaction of a mild or even mildly serious nature might take place in the first six to twelve hours, is, I think, an apt hypothesis. That the mobilization of certain chemical substances follows operative procedures on the large bowel and that this is taken care of by the blood transfusions, is another theory which I am not able to debate, but the confirmed opinion that routine transfusions is distinctly desirable following colonic and rectal resections, is definitely borne out in my experience.

Table III indicates the trend in my service toward the one stage operation during the past three years. Clearly, however, there is an indication for an occasional two stage operation which embodies the features of the one stage radical procedure. Again, there is a very definite proportion of cases which cannot be submitted to either of the formidable combined procedures but which can be operated upon by the colostomy and posterior type of operation of Mummery.

Table III

CANCER OF THE RECTUM AND RECTOSIGMOID

50 Cases

Resections	38
Explorations	12
Operability	76%

TYPES OF RESECTION

One stage combined abdominoperineal (Miles)	—18
Colostomy and posterior (Mummery)	—16
Two stage combined abdominoperineal (Rankin)	— 4

MORTALITY

Mortality—Total group (50 cases)	5
Following colostomy and posterior resection	1
Following colostomy alone	4
Causes of death—Pulmonary embolus	2
Coronary occlusion	1
Peritonitis	1
Intestinal obstruction (acute)	1

CONCLUSIONS

It has seemed advantageous to me in the past three years to make the following changes in the handling of cases of cancer of the rectum and rectosigmoid: first, the abandonment of intraperitoneal vaccination as a preliminary preparatory step (the elimination of vaccination has been done with regret and with the hope that some utilizable step in this direction may be found in the future); second, the elimination of spinal anesthesia as a routine anesthetic; third, an extension of the period of preparation to at least seven days; fourth, the routine performance of a presacral neurectomy; fifth, postoperative blood transfusions; and sixth, a wider use of the single stage abdominoperineal resection after the technic of Miles.

Even though the ideal operation is a radical resection in one stage, there is small question that a definite field for a radical procedure by graded maneuvers exists. Unquestionably, there are cases which are considered borderline for surgery which may be advantageously operated upon by a two stage radical maneuver, thus increasing the operability percentage without increasing the mortality statistics. Furthermore, the conclusion is inescapable that there are many cases upon whom it is impossible to employ either of these radical procedures and yet in whom resection may be undertaken by a less formidable operation, namely, colostomy and posterior resection.

With the statistical data available to prove that from 38 to 79 per cent of cases of cancer of the rectum and rectosigmoid may be given a chance of five year cure by the utilization of either the one or two stage combined operation, or the less radical colostomy and posterior resection type of maneu-

ver, the hopeful prognosis following surgery for cancer in this location is definitely established and the indications for the different types of surgical operation are rapidly becoming more and more clearly recognized.

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THE ONE STAGE PROCEDURE OF THE TREATMENT OF CARCINOMA OF THE RECTUM

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FOR many years carcinoma of the rectum was treated in this clinic by a variety of methods, depending upon the opinions of a number of different surgeons. The results were variable but in the main unsatisfactory. The operative procedures directed to a local removal had a low operative mortality but also a very low incidence of cure, while the more extensive operative procedures had a high operative mortality but a higher percentage of cures. On considering the situation it was obvious that we were learning slowly, and by bitter experience, a lesson that had been taught clearly and with finality by Miles.[1] His brilliant and painstaking investigation of the lymphatics of the rectum, his observations on the mode of spread of carcinoma of the rectum, and his study of end-results of all types of operative attack were so conclusive that we felt it imperative to base our treatment on his conclusions.

From 1931 to 1936 we have endeavored to follow his teachings and methods in treating carcinoma of the rectum and it is with our personal experience during this time that we wish to deal here. No two clinics have the same cancer problem, which varies with the social and economic status of the patients treated in that clinic. In the University Hospital, the majority of the patients come when they are incapacitated, which means that, by and large, patients with cancer are seen here late in the course of the disease.

During this period we have had the opportunity of studying 270 patients with carcinoma of the rectum or rectosigmoid. Of these, 183 were men and 89 women. Forty-six patients came for diagnosis only, or refused operation, or were treated elsewhere, leaving 224 patients receiving treatment in this hospital. The age distribution of the patients corresponds with that of other cancer groups. The diagnosis was proven in every case by examination of biopsy or operative specimen and the tumor types correspond to the usual findings in this group of patients. After examination it was found that 114, or 51 per cent, of those entering the hospital for treatment were unsuitable for radical operation because of the far advanced lesion or because of associated disease. An analysis of the treatment carried out for these patients is shown in Table I.

Some in the terminal stages of the carcinoma, often with widespread metastases or with advanced other disease were not given any treatment for the carcinoma. Another small group was treated by roentgen therapy, radium or electrocoagulation; this includes patients with squamous cell carcinoma

of the anal canal, for whom we advise irradiation therapy. Palliative colostomy was performed on many for the reasons shown in Table II.

TABLE I

PATIENTS UPON WHOM RADICAL OPERATION WAS NOT ATTEMPTED

Unsuitable for radical operation		114 = 51%
No treatment advised		
Terminal stage with widespread metastases	12	
Far advanced other disease	4	
Palliative operation		
Colostomy	75	
Colostomy before entrance	10	
Radium and roentgen therapy	2	
Electrocoagulation	2	
Cystostomy	2	

TABLE II

REASONS FOR PALLIATIVE COLOSTOMY ONLY

Performed elsewhere		10
Colostomy in hospital		75
(a) Local extension to peritoneum, bladder, prostate, vagina, fistulæ	43	
(b) Local lesion operable but metastases in liver	11	
(c) Locally inoperable with liver metastases	15	
(d) Metastases in inguinal nodes	1	
(e) Age	5	

The largest number were unsuitable for radical operation because of local extension of the carcinoma to the viscera lying adjacent to the rectum. Direct infiltration of the bladder, uterus, broad ligaments, vagina or prostate, or infiltration of the peritoneum of the pelvic floor, rectovesical or vaginal fistulae, were all regarded as hopeless. This type of extension prevented resection in 43 instances in all of which the liver was free from palpable metastases. In 11 patients, the local lesion seemed suitable for radical operation but the liver was grossly involved with metastases, while in 15 instances the lesion was hopeless both because of local extension and because of metastases in the liver. Positive evidence of metastases in the inguinal lymph nodes precluded resection in one case while old age made resection impossible in five patients in whom the lesion was otherwise suitable. We feel that in none of these patients was a favorable chance for radical operative attack on the carcinoma missed.

The hospital mortality of these patients upon whom colostomy was performed was under these circumstances 22 per cent. Of these, more than one-half, or 12 per cent, of the group died within the first week, while the remainder lingered on to die after an average of 35 days, of some complication of the original disease. We have been interested in following this group of patients in an endeavor to see whether colostomy is worth while in the hopeless cases (Table III).

Table III

MORTALITY IN PATIENTS UPON WHOM PALLIATIVE COLOSTOMY WAS PERFORMED

Total number palliative colostomies		85
Hospital deaths		18 = 22%
(A) Died within 14 days	10 (avg. 6 da.)	= 12%
(B) Lived over 14 days	8 (avg. 35 da.)	= 10%
Cause of death in Group A:		
Intestinal obstruction	4	
Pneumonia	1	
Peritonitis	1	
Cardiac disease	1	
Age, disease	3	
Group B:		
Died of extensive cancer, general peritoneal, liver.		

If the patients were able to leave the hospital, they were found to live an average of ten months. Whether this is worth while it is difficult to say, but there is usually a marked lessening of pain, bleeding, and tenesmus, and about one-third of them gained weight and strength. The shortest duration of life was one month, while one patient, with proven carcinoma, is still alive after four years.

From this rather brief experience we feel strongly that in the patient unsuitable for radical operation, a colostomy is worth while and that the earlier in the course of the disease it is performed, the more beneficent are its results. There is a common feeling that this operation should only be carried out for obstructing lesions. This view is fallacious, as it gives as much comfort to those suffering from pain, tenesmus, and hemorrhage as it does to those with obstruction. Obstruction usually develops late, or never, and to wait for this complication to ensue is to deprive the patient of much comfort and to shorten his life. Colostomy performed in the terminal stages of the disease carries a high mortality, when its utility is questionable. In the majority of patients in whom for one reason or another one decides to employ roentgen therapy or radium, we have found it more comfortable and in general more satisfactory to carry out this treatment after a colostomy has been established.

Obviously no one type of operation should be used for all kinds and locations of carcinoma in any organ and we have endeavored to suit the treatment to the lesion. Seven patients with small, low lying lesions were operated upon by preliminary colostomy with a later perineal excision. Three of these patients were also regarded as poor risks for a more extensive operation. The single death in this group occurred suddenly ten days after the second stage, in a patient having a normal convalescence. Unfortunately, an autopsy was not permitted.

The abdominoperineal operation was carried out in two stages on 27 patients and in one stage on 72 patients. We feel that the greatest contribution to the success of this operation, no matter how it be carried out, is the principle of proper rehabilitation of the patient so strongly insisted upon

by Miles in England and Rankin[2] in this country. In the earlier part of this period, we employed a two stage procedure as a routine with results that were satisfactory to us. The operation commonly used was that of exploration through an inguinal incision and if found satisfactory for the radical operation, the sigmoid was cut across, the lower end being closed and dropped into the abdomen while the upper end was drawn out through the incision to form the permanent colostomy. Later, the lower end of the sigmoid and the rectum were removed at the second stage. It appeared that the second stage of this operation was as long and as difficult as the one stage operation and the mortality of the one stage procedure should not be higher because of operative trauma if the patient could be brought to operation in as good condition. Often the second stage of the two stage operation was technically more difficult than an original attack would have been, since adhesions were frequently found that hindered a smooth dissection. It was then decided to attempt the one stage operation after the method of Miles upon a series of patients, on the assumption that it would be an easier operation to perform, it might shorten the patient's hospitalization and, at least, we would learn of its limitations.

The methods of preparation of the patient developed by other workers in this field have been employed. Usually from five to ten days are needed for this process and the average time spent in the hospital in this group was seven days. The bowel is emptied by the use of weak saline purges given frequently, with daily enemata. Most patients with mild symptoms of obstruction can be decompressed satisfactorily by this regimen. Cecostomy was necessary in only three patients, all others being handled by nonoperative measures. It is probable that we have erred on the side of not giving enough time to the preparation and in the future we purpose to take a few days longer for this important step. The patient should spend most of the time in bed, eating a low residue, high caloric diet with high fluid intake. Associated defects are corrected and transfusions are given in case anemia exists.

In all of the patients upon whom the one stage operation has been performed the peritoneum has been protected against infection by the introduction of bactrogen as developed by Steinberg.[3] If the operation is performed in two stages, the preliminary colostomy has the same effect of raising the local immunity of the peritoneum to infection and therefore the introduction of any substance for this purpose is unnecessary. This substance is composed of 5 per cent aleuronat, 600 million B. coli, killed by formaldehyde, and 30 cc. of 1.5 per cent solution of gum tragacanth. In the early cases this was introduced into the peritoneal cavity 48 hours before operation but this time has been shortened until it was given 12 hours before operation. Steinberg has recently found that the desired reaction can be secured in three hours and we now introduce the bactrogen at the close of the operation. The effect produced is not specific but is entirely an hyperleukocytosis. The leukocytes will attack any bacteria that are capable of being destroyed by phagocytosis. The important point is to produce a sufficiently large number of phagocytes, in a short period of time, to destroy

bacteria present before toxins can be developed. The action of the bactrogen is limited in time and after three days the protective reaction rapidly disappears. It can be reintroduced at this time if it is desired to maintain an hyperleukocytosis as a barrier against infection. We feel that the use of this substance is worth while to guard against contamination that occurs not infrequently in the operations we have performed. In dissecting the rectum, in those cases in which the carcinoma has infiltrated the entire thickness of the bowel, it is almost inevitable, at times, that the bowel may rupture with resultant contamination of the field. This has occurred 11 times in this series, and while the wound in the abdominal wall and the perineal wound have been infected, the peritoneum has apparently taken care of itself in every instance. Sepsis may later spread from the infected posterior wound but in no instance have we seen acute peritonitis of the usual type follow even gross contamination when the bactrogen was used. It would seem that this protection of the peritoneum is worth while in the one stage procedure as at least giving the same immunity to infection that it has in the two stage operation as a result of the carrying out of the preliminary colostomy. We have used the intraperitoneal injection of some form of bactrogen about 300 times and no harmful effects have been noted. There is usually a fever that averages 101° F. and a general leukocytosis that averages 17,000. There is some abdominal discomfort that is readily controlled by an opiate.

The anesthetic employed has uniformly been spinal, supplemented if necessary with very light nitrous oxide and oxygen. It has been found that a comparatively small amount of drug is adequate and the usual dose is 80 mg. of novocaine to which in the past year we have added 20 mg. of pantocaine. The effect of spinal anesthesia on the bowel makes the abdominal part of the operation very much easier and is almost a necessity.

The operation is carried out as closely as possible following the principles of Miles. With the patient in the Trendelenburg position a three inch incision is made through the inner third of the left rectus muscle and exploration carried out. If the lesion is not suitable for radical removal a colostomy is performed through this incision. If the radical operation is feasible the incision is enlarged to provide a good exposure and the operation carried out as described by Miles. The upper end of the pelvic colon is either drawn out through a stab wound in the left inguinal region or allowed to project from the original incision. No sutures are placed in the bowel which is allowed to remain without tension where it will. It is desirable to leave as much bowel out as can be readily secured, since it is impossible to predict exactly where the blood supply will be and a long segment of exteriorized bowel will enable one to guard the wound against contamination with some certainty. As soon as the wound has healed, the protruding bowel is cut across about two inches from the skin surface to form the permanent colostomy. The patient is then placed in the Sims position and the rectum dissected out with the fat from the ischiorectal fossae. The prone position with hips flexed was used on many of the patients as it gives

a much better exposure for the surgeon but it was noticed that there was frequently a fall in blood pressure with this change of position and consequently it has been given up for the lateral position, which gives a satisfactory exposure, and in which no changes of blood pressure have been noted. The posterior wound is closed at both ends leaving the middle wide open for the introduction of a large sheet of rubber dam which is passed well up to the pelvic diaphragm. This is removed in three or four days and replaced by smaller drains of the same material.

Following the operation a transfusion is given if the patient shows any signs of shock or if he fails to react in a perfectly satisfactory manner. Transfusion has been carried out in about one-half of the patients in this group.

The postoperative care of these patients has no special features except the handling of the colostomy. One of the outstanding advantages of the two stage operation is that the colostomy is established and working while in the one stage procedure the difficulties of regulating the colostomy are superimposed upon a patient recovering from a severe operation. We have not had any great difficulty with the colostomy since we have regarded it as a true intestinal obstruction. The exteriorized loop is left closed as long as possible without allowing abdominal distention to occur. A catheter may then be introduced which often will care for the obstruction for several days longer. Eventually the loop is completely opened. We have discontinued the use of irrigations or any other source of irritation of the colon. If reverse peristalsis occurs with nausea, vomiting or distention, constant suction of the stomach, after the method of Wangensteen, is employed. This procedure is used intermittently until the tone of the intestinal musculature is restored and normal peristalsis is reëstablished.

The patients are kept in bed for two weeks and then allowed up for increasing periods of time. They are urged to walk as soon as they are able as it seems true that the posterior wound fills up much faster if the patients are up and about. The average time of hospitalization after operation is 27 days. The posterior wounds are healed in about three months.

Results.—The hospital mortality in all patients upon whom an attempt was made to cure by operative measures is shown in Table IV, but these figures taken alone do not make a true commentary on the actual facts.

Table IV

CASES UPON WHOM CURATIVE OPERATIONS WERE ATTEMPTED

	No.	Mortality	Per Cent Mortality
Colostomy—did not return for second stage	4		
Colostomy with perineal excision	7	1	14
Two stage abdominoperineal operation	27	7	26
One stage abdominoperineal operation	72	12	16.5
Total	110		

The mortality for the two stage operation is 26 per cent but this is unfair to this procedure. Twenty-three patients considered as good subjects for radical operation were operated upon by this method with a loss of three patients as shown in Table V, a mortality of 13 per cent.

TABLE V

PATIENTS DYING FOLLOWING MULTIPLE STAGE ABDOMINOPERINEAL OPERATION

	Age	Sex	Time	Cause	Remarks
(1)	64	F	45 days	Sepsis. Perineal with fecal fistula in perineum	Favorable lesion
(2)	45	F	11 days	Peritonitis. Wound sepsis. Hepatic metastases	Local lesion favorable
(3)	69	F	2 days	Peritonitis. Pneumonia. Wound sepsis	Hypertension. Lesion favorable
(4)	55	F	1 day	Shock	Advanced lesion infiltrating vagina, supravaginal hysterectomy also done at time of operation
(5)	48	M	1 day	Cardiac collapse	Extensive lesion. Could not be removed completely
(6)	64	F	8 days	Auricular fibrillation. Cardiac failure	Hypertension ¾ annular. Nodes +
(7)	63	M	8 days	Auricular fibrillation. Cardiac failure. Anuria	Favorable local lesion

Within the past year four patients were operated upon in stages because they were recognized as desperate risks and it was hoped that after a colostomy and a rest period of several weeks, they might be generally improved to a point where a cure might be attempted. Two of them were elderly people with marked hypertension and cardiovascular disease and the other two were patients with far advanced lesions fixed in the pelvis. Eventually a radical resection was attempted with fatal results in all four patients. Consequently the mortality is not that of the operation but was due to the poor judgment that attempted operation on patients that were inoperable.

As shown in Table IV, the hospital mortality for the one stage procedure was 16.5 per cent, which requires some explanation. In the first 48 patients upon whom this operation was performed, there was a mortality of 8.3 per cent. These patients were carefully selected both from the standpoint of the lesion and their general condition. During the past year we have striven to increase the operability of our patients with carcinoma of the rectum, which meant that we have operated upon patients who are not good risks because of advanced age or other general conditions and we have attempted operation upon lesions that have been found to be locally hopeless when operation was finally attempted. The four patients already mentioned as dying after the two stage operation fall into this group. In Table VI is shown a more detailed analysis of the patients dying after the one stage

procedure. The first four patients were old, and poor risks on that account. All of them lingered on for from nine to 17 days, dying of pneumonia or cardiovascular disease. Only one patient in the group over 70 survived the operation and it is questionable whether one should attempt the operation in anyone past 65.

TABLE VI

PATIENTS DYING FOLLOWING ONE STAGE ABDOMINOPERINEAL OPERATION

Patient	Age	Cause of Death	Time	Remarks
(1) B. F.	74	Pneumonia	9 days	Advanced lesion invading pelvic fascia
(2) N. H.	71	Auricular fibrillation. Uremia	17 days	Obesity. Local lesion favorable. Nodes +
(3) G. M.	72	Auricular flutter	17 days	Local lesion favorable
(4) A. S.	66	Bronchopneumonia	14 days	Local lesion favorable. Advanced arteriosclerosis
(5) C. D.	55	Pneumonia	2 days	Advanced lesion invading pelvic fascia. Hypertension. Chronic nephritis. Liver metastases at autopsy. Nodes +
(6) M.W.	36	Obstruction. Infection in posterior wound	14 days	Far advanced lesion infiltrating. Nodes +
(7) W. O.	65	Sudden death. ? vascular accident	2 days	Advanced infiltrating lesion. Nodes +. Obesity
(8) H. L.	60	Gangrene colostomy loop. Reoperation	6 days	Advanced infiltrating lesion. Nodes +
(9) B. T.	58	Posterior wound sepsis. Secondary hemorrhage	9 days	Favorable lesion
(10) E. S.	43	Pneumonia-embolic. Septicopyemia. Posterior wound sepsis	24 days	Local lesion favorable
(11) P. B.	52	Posterior wound sepsis. Urinary tract infection. Peritonitis	35 days	Lesion favorable. Nodes +. Obesity
(12) W. L.	65	Bronchopneumonia	2 days	Favorable lesion. Moderate arteriosclerosis

The next four patients all had lesions that had passed through the bowel wall and invaded surrounding structures and viscera. In short, they had lesions incurable by operation and operation should not have been attempted. The operative mortality is high in these patients as anatomic planes are lost, operative trauma is greater, and infection, by breaking through the bowel, is common. The first eight patients in the mortality list were all badly selected as only two of them had lesions that might have been cured by operation and these two patients were poor operative risks because of advanced age.

There were two deaths from postoperative accidents, one from hemorrhage from the posterior wound on the ninth day and another from sloughing of the exteriorized bowel. Three other patients died of infection, two from extension of infection from the posterior wound and one from pneumonia. Of all these patients only six had lesions favorable for operation. The mortality for the one stage operation is then somewhat, if not largely, dependent upon the selection of patients for the operation.

In our endeavor to increase the operability of patients with this disease we have certainly operated upon patients who should not have been treated in this manner. While we have attempted curative operations in 49 per cent of the patients, the true operability was probably not greater than 35 per cent. The mortality has risen as we have increased the percentage of those operated upon. One should be critical of high operative mortality but in this instance not too critical, as we are dealing with patients with hopeless lesions that will in a short time cause death anyway, and if, by an aggressive attitude of mind, one can cure an occasional borderline case, it may be worth while. We have determined, however, to return to a somewhat more conservative selection of patients for the radical operation. Every patient over 65 should be considered most carefully before advising operation. Patients with marked hypertension and cardiovascular disease should probably all be treated conservatively. The matter of local extension of the lesion to pelvic structures, as shown by fixation, is often hard to determine, but clearly we have tried to resect a number that were not resectable and our judgment here has most often been at fault. With a better selection of patients, the mortality of the radical operation will be lower. From this rather limited experience, we feel that the one stage procedure has a distinct place in the treatment of carcinoma of the rectum and rectosigmoid. If obstruction is not present and if plenty of time is taken to prepare the patient for operation, we feel that the mortality from the operation should be not greater than that of the two stage operation. The greatest mortality, with us, has been due to a deliberate attempt to increase the operability of the disease and is not due to the use of any particular type of operation. With a proper selection of patients, the mortality should be 10 per cent or less.

CONCLUSIONS

The most important point in the operative treatment of carcinoma of the rectum is the recognition of the importance of the radical method of abdominoperineal resection as advocated by Miles.

Whether this is accomplished in one or in several stages is a matter of personal preference of the surgeon and of distinctly less importance. Most patients can be operated upon as safely by the one stage as with multiple stage operations if proper preparation of the patient is effected.

Higher operability will come with earlier diagnosis and not from attempting the operation upon far advanced lesions or on the poor risk patient.

Bactrogen (Steinberg) has a place in protecting against infection if gross contamination occurs.

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DISCUSSION OF THE PAPERS OF DOCTORS RANKIN AND COLLER

Discussion.—Dr. Daniel Fiske Jones (Boston, Mass.).—I believe that a two stage operation, or the posterior operation of Mummery, should be the operation elected by those of little experience. And I think that if one pays attention to these cases he will find that the number of one stage operations will increase quite steadily. I have been accused of performing the two stage operation always; as a matter of fact I have performed just three two stage operations in the last two years, which I think corresponds very well with what Doctor Rankin has said.

It seems to me to be our duty to operate upon these patients when possible, and one cannot operate upon all of them by a one stage procedure. I cannot understand Dr. T. E. Jones' attitude toward the two stage operation. In the most recent article of his, that I have seen, he states that if you perform a two stage operation there are mortality risks incident to both stages, and that the mortality in those two stages will probably be equal to, if not greater than, the mortality in a one stage operation. That depends upon what you have been doing and what you want to do. I know that there are some patients upon whom I have operated with a two stage operation or with a posterior operation, who could not possibly have been operated upon with a one stage operation. It is, I believe, much better to perform a two stage operation, either a combined abdominoperineal amputation in two stages or a colostomy and posterior excision, than to do nothing at all. I therefore do not see that Doctor Jones has any argument at all, and I do not know why he should just throw out the two stage operation.

I have never in any of my articles, that I know of, stated that you must do a two stage operation or that you must do a one stage operation. The man who is doing this work must have some judgment as to what to do, and I would advise the surgeon beginning with this operation to begin with a Mummery operation, namely, a colostomy and posterior excision, or a two stage combined abdominoperineal operation, and then go on to the one stage operation as he feels that he has the ability to do it. If he does that, he will save a good many lives.

As to vaccination, I have not used vaccination for one particular reason, and that is that if you have a colostomy which you want to close or a colostomy following a Mikulicz operation, there is no use attempting to close it inside of eight weeks, and the nearer you are to the time of the first operation, the surer you are to have infection. It seems to me, therefore, to be a rather useless procedure to use the vaccines and then go to work and operate within a week. It takes at least six weeks for a patient to be-

come immunized after a colostomy; it would seem reasonable, then, to assume that it would take more than 24 hours or a week to immunize a patient with vaccines.

As to the resection of the presacral nerve, I wish that Doctor Rankin would find a method of improving the bladder function afterwards. Mr. Miles nearly floored me when he told me that he had never had a cystitis following an operation for carcinoma of the rectum. I have never had anything but temporary paralysis and cystitis. I began, and have continued up until just recently, with the removal of the presacral nerve. Now I am not resecting it, but I cannot see that it makes an iota of difference. If Doctor Rankin can, I shall try it again. I do get infection of the bladder and I do not see how you are going to avoid it as a resultant of the temporary paralysis.

As to the mortality rate, there is no use talking about the mortality rate in these cases unless you give the percentage of operability. Doctor Rankin has just said that his operability was 76 per cent. He must have a large number of good general practitioners down around Lexington, because I have never been able to approach 76 per cent. I have been up to 67 per cent, but that included the posterior operation and the two stage operation.

One thing that I should like to do is to urge every man here to remove the growth when possible, whether there is a nodule in the liver or not. The condition of these patients following removal of the growth and a colostomy cannot be compared in any way with the physical discomforts, displeasures and the mental effect which follow a simple colostomy. I think that if any one of you had a cancer of the rectum and somebody told you that he had performed a colostomy for you, you would not get very much pleasure out of that operation if you knew that the growth had not been removed. On the other hand, if a surgeon can go to a patient and say, "Yes, I have made a colostomy as I said I would, but I have removed the growth," the patient will have a good time so long as he is comfortable. And I believe that in these cases that is worth while.

As to the interest in these cases, I am pleased that there is very little talk now about not being able to perform the operation because the patient would have to have a colostomy. That attitude, thank goodness, has largely disappeared.

Doctor Rankin read a paper at the Southern Medical Association meeting and told them that they should perform colostomies even as palliative operations, and there was not a murmur against it. The last time I urged colostomies in all cases of carcinoma of the rectum, I was set upon by several older gentlemen who told me that they had operated upon cases who were alive and well after 14 or more years. I asked, "What operation did you do?" They replied, "Just a local operation." That was to show me that the colostomy was quite unnecessary. It really is a great pleasure to see the change in the attitude of the doctors around this country in regard to a colostomy.

If I may say something about a colostomy, I should like to state that there is no such thing as control of the colostomy by any operation that I know of, and I should be very pleased to know if anybody here has ever heard of a type of colostomy that would control the bowel movements, because I should like to try it. I have never seen one yet. You can control the evacuations, however, by teaching the patient what and how to eat and how to take care of his bowels. I have very, very few patients who ever have any trouble with their colostomies after being taught how to care for

them. If they try to care for their colostomies themselves without instructions, they always make a mess of it.

If it would be of any comfort to some of you gentlemen, I should like to say that while Doctor Coller keeps his colostomies closed as long as he can, I think that he will get along with perfectly clean wounds in 99 per cent of his cases if the bowel is brought out of the original abdominal wound and if the colostomy is opened within 24 hours. I think that if you open up your colostomy early, you are not liable to have your patient nearly so distended and uncomfortable, and the wound will not become infected.

Dr. Frank H. Lahey (Boston, Mass.).—I think that carcinoma of the rectum is such a hopeful lesion, when all of us can report five year nonrecurrences ranging, we will say, from 35 to 45 per cent, that this subject demands a great deal of interest. It demands that all of us interest ourselves in the various measures which will increase operability, lower mortality and increase curability.

As to the two stage and one stage procedure, it seems to me that all of us owe a great deal to Doctors Mummery, Jones, Rankin, Lawrence Abel, Thomas Jones, and others who are employing these different types of procedures. But we come back to one thing that I would urge as the result of our experience, and that is that you can only learn by experience with these cases, and that the two stage procedure in the hands of the men who have not had a large experience is certainly safer than a widening of the range of one stage operations before you have had a large experience. It is like preliminary ligations of the thyroid that we spoke of yesterday and two stage thyroidectomy. You cannot write such descriptions down; you cannot vividly describe such cases and indications. If you could, you could take a correspondence course in when to do the two stage operation and when to do the one stage procedure. I think that there is nothing which has been more strikingly demonstrated to us than the need for experience before forsaking two stage procedures. None of us desire, particularly, to discuss types of operation. We all do our own operation better than the other fellow's; we all select the type of operation that fits us and fits our cases.

I think there are one or two things that are of interest. Our operability is the same as Doctor Rankin's and I think it can be kept that high. It certainly has definitely increased. Up to 1934 it was 53 per cent, and it has been raised to 73 per cent during the last two years. Our mortality, when operability was 53 per cent, was 8.25 per cent, and now it is about 12 per cent.

I agree with Doctor Jones. I do not think we ought to be too much interested in mortality, because, as Doctor Rankin has said, when you get interested in mortality down comes the operability, and down comes the curability. I agree also very definitely with Doctor Jones that the primary consideration is to make these patients live the longest time comfortably and that is the reason we have increased our operability.

There are one or two other points which I think are of interest and value to anyone who is dealing with cancer of the rectum; one is to aggressively oppose the patient and the general practitioner who want to limit the operative procedure because the lesion is small. So many times these patients are sent to us by their doctors with a small lesion the size of my little fingernail and they want a local resection. If ever there is a chance in the world to accomplish cure, and if ever there should be a time when aggressive surgery should be undertaken, it is then. There are so many sad experiences. A few years ago a young man with a papilloma just within the anus came to

us, rejected radical operation and had local application of radium elsewhere, only to return in a year with a recurrence, have us do a two stage operation and die of the recurrence. That boy certainly should have had a radical operation in the beginning and if he had would probably be alive today. I think it is our duty to advocate radical surgery in the presence of small lesions.

I think we owe Doctor Jones a great deal for stressing colostomy. The patient judges the end-results in terms of his colostomy. He forgets within a year that he has been cured or relieved of a carcinoma and thinks only in terms of how well he gets on with his colostomy.

There are certain things concerning colostomy which from our experience are also of value. If you want to have good results from colostomies, make the patients come back frequently, see them frequently, listen to their problems and teach them how to constipate themselves. There is something about a colostomy that takes time for adjustment. Warn the patient that for six months he will have a little trouble about managing his colostomy, and at the end of that time he will begin to know how to manage it, and he and it will function better. Place, as we have, in various parts of the community people who are grateful for relief and who manage their colostomies well. They will go out and help convince individuals who reject radical operations by demonstrating that their colostomies do work well. I believe this is an extremely hopeful lesion, that we should all approach it aggressively and enthusiastically and that with this approach we can widen the operability, decrease the mortality and increase the curability.

Dr. Mont Rogers Reid (Cincinnati, Ohio).—I want to bring up one point which was not touched upon by either Doctor Rankin or Doctor Coller. A few years ago I advocated the judicious use of lead and opium pre- and postoperatively, when dealing with carcinoma of the large bowel. This has proved of the greatest assistance. After the patient has been properly prepared and the intestines cleared out as well as possible, the lead and opium are given a day or two before the operation, and then postoperatively as long as one wishes to prevent any bowel movements. Under this regimen one can make the colostomy opening at any time and get absolutely no fecal drainage. It does, however, allow the escape of gas. In addition its use has another distinct advantage, in putting the intestines at rest, which materially aids in wound healing. I never resect intestines without the use of lead and opium both before and after operation.

Dr. J. Shelton Horsley (Richmond, Va.).—There are two points that I should like to discuss briefly. One is concerning the vaccine, or more properly, the coli-bactragen of Steinberg, that Doctor Coller referred to. I have been using it for some time. My conclusions have been somewhat different from his. I noticed that several times there was a very severe reaction. On one or two occasions I have been forced to postpone the radical operation because of the illness of the patient. Steinberg says that the height of immunity is about 48 hours, but these patients are too ill, and invariably when I have postponed it for, say, six or seven days, contrary to experimental results, the results have been rather happy, and almost always when I have operated in the first 48 hours the results have not been so good.

In regard to the resection of the rectosigmoid or the upper rectum, I have employed a procedure that was apparently quite satisfactory, so far as immediate results are concerned. In the upper rectum and the terminal sigmoid, it is the custom, I believe, to make a permanent colostomy. If, however, the patient is prepared by a competent cecostomy on the right side,

through a muscle-splitting incision, in which the bowel is brought up and a glass rod is put through the ascending mesocolon, so that the distal bowel can be put at complete rest and irrigated with salt solution either through the colostomy or the rectum once or twice a day, and the coli-bactragen is given, an end-to-end union can be effected. The patient is placed in the extreme Trendelenburg position. The operation is performed as if the entire rectum were to be extirpated, and the tissues in the hollow of the sacrum are freed as in the complete radical operation. The upper stump is then brought down after it is cleaned by placing two long tractor sutures of linen, after rotating the upper stump outward so that its posterior surface will be covered with peritoneum. These very long sutures are gradually tightened as the upper stump is shoved down into the pelvis. The tractor sutures are then tied, and the posterior margin of the upper stump is sutured to the posterior margin of the lower stump, making it quite snug. This suture is carried along in front, inverting the margins as much as possible. This row of sutures is reinforced by a series of interrupted mattress sutures of fine chromic catgut which are then passed through adjacent fat.

Two weeks later, the enterostomy is closed. Enemas should never be given after the resection. I am confident I killed a patient by administering one two weeks after a resection, when he was doing pretty well.

That procedure, I believe, will, in that type of case, substitute an end-to-end union for a permanent artificial anus, and effect a sufficiently radical operation.

Dr. Harvey B. Stone (Baltimore, Md.).—I think both of the essayists are to be congratulated for their courage and honesty. Of particular interest is the statement by Doctor Rankin that he has come to the conclusion that the vaccine which he did so much to develop, and in which he was so hopeful of benefit, has, in his judgment, no longer sufficient justification to be continued, and the frank admission by Doctor Coller that the effort he made to extend the operability in his series of cases seems unwarranted and that he is going to reverse his position.

I should like to say, too, that I agree with all six of the modifications that Doctor Rankin has made lately in his technic in the handling of these cases. They coincide very closely with our practice in Baltimore, and we have felt that they are all helpful.

There is one phase of the subject which I would like to call attention to: A question of certain types of inoperable carcinoma of the rectum or rectosigmoid, where operative attack as the best form of palliation, as advocated by Doctor Jones, seems too extensive a procedure to be justified; where, for instance, the bladder, or other adjacent structures, are involved, and the palliative removal of a hopeless carcinoma is too formidable an operation to be worth attempting, and yet the patient is not actually obstructed.

There is a procedure which has given us a good deal of help at times, and that is the performance of a so called precolostomy. In such cases, after the exploration has determined that it is inadvisable to make an attempt at removal of the growth, and at the same time the patient is not seriously involved in difficulties of an obstructive nature, we draw up the sigmoid into a small lateral wound and fix it there without opening it, fixing it in such a way that only a small portion of its circumference is attached to the skin.

That forms a potential colostomy, which may be opened as desired at any later period. In a good many such cases patients may not die for a year or more subsequent to the exploratory operation, without ever requir-

ing the colostomy to be opened. On the other hand, some cases following the handling of the exploratory procedure, within a few days after the first operation, develop complete obstructive conditions, and at once, without further difficulty, the knife can be plunged into the attached loop of bowel and the colostomy completed.

Dr. Henry F. Graham (Brooklyn, N. Y.).—We have listened to masters of surgery, men who are experts in the surgery of carcinoma of the rectum. It might be of passing interest to learn of the reactions of an ordinary, everyday surgeon, not an expert.

I have seen both Doctor Rankin and Doctor Lahey operate. As a casual operator, I have seen some 75 cases personally in the last 25 years. I do not know my mortality, but I am sure it is high. Since I have learned how to do the Lahey operations (it took me two years to perfect myself in the different steps so I could perform them properly), I have rather ceased to dread these operations.

I have had several experiences which convinced me, at least, that for the ordinary surgeon it is the safest procedure that can be employed, especially in such cases as have been mentioned where the growth has progressed through the entire thickness of the bowel wall and has invaded the surrounding tissue.

The Lahey operation gives one an opportunity to use mercurochrome, or other similar material, in the lower loop. In one case, at least, I twice broke through into the lumen of the bowel during the removal of the lower loop, and no infection resulted, either in the lower wound, or in the peritoneal cavity.

At the present time we have a case of carcinoma of the rectum, who was operated upon by my associate. I do not think he had ever removed a carcinoma of the rectum before. We considered the case carefully in all its aspects. As a result, this patient has been through both steps of this operation and is now convalescing without incident.

Dr. Frederick A. Coller (Ann Arbor, Michigan).—I have just one comment to make, and that is, that I still think the hyperleukocytosis that is produced by Steinberg's bactrogen is worth while in many instances. It is not a vaccination in any way, but simply produces an irritation with a marked local leukocytosis, and I may say, for Doctor Horsley's benefit, that it has been changed tremendously within the past year. The action is now obtained in three hours, and the reaction (we have used this form on 50 or 60 cases) is minimal. If contamination does take place, as it will not infrequently, one has in this, I think, a substance that is of distinct benefit.

I have tried to bring out the fact that, in the patients that we treated in this group which has been analyzed, we have positive proof by autopsy or direct observation that 68 and perhaps 70 per cent of them were hopeless, and that the lesion had passed beyond the reach of any surgeon. Higher operability will come with early diagnosis. The patients coming to our clinic now come, in most instances, because they are incapacitated, consequently our operability is low. We have tried to increase the operability by attempting to cure hopeless cases. We have learned our lesson and will try in the future to increase the percentage of operability by teaching earlier diagnosis.

IMPERFORATE ANUS WITH RECTOVAGINAL CLOACA

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The various anomalies in the development of the rectum and anus are well known to the student of embryology and of interest to the surgeon. In some of them complete obstruction of the bowel exists and requires prompt intervention to save the infant's life. In others the rectum is not completely blind, but instead of opening through the anus normally, the anus is absent and the rectum opens into some other hollow viscus. This paper concerns itself particularly with those cases in the female in which the rectum opens into the vagina. These organs normally communicate or form a common cloaca during one period of embryonic development, but later the hindgut becomes separated from the urogenital sinus by the development of a septum between these two passages, and the former, the hindgut, opens to the surface by its fusion with the proctodeum that dips inward from the perineum, thus leading to the formation of the normal anus. Sometimes both these processes, that is, the separation of rectum from vagina and the opening of proctodeum into rectum, fail to occur and the incomplete embryonic state persists. This leads to a condition of imperforate anus and rectovaginal fistula or cloaca.

The occurrence of such an anomaly has been long known. Bodenhamer,[1] in his interesting monograph entirely devoted to congenital anomalies of the rectum and anus, cites references dating back to classic antiquity and mentions records of similar lesions in the dog and cow. He states, however, that this is a rare condition, and cites all of the few instances to which he could find reference. Since his time there have, of course, been other cases reported, but one is struck by the scantiness of discussion of the subject in any of the works consulted. Many of the text-books on surgery omit mention of it or barely state that such an abnormality exists. Even with volumes devoted to the rectum and anus the subject is dealt with, if at all, in very cursory fashion. This is, of course, entirely proper. The rarity of the condition does not justify extensive discussion in general treatises. Furthermore, as several authorities point out, this particular form of imperforate anus is apt to be less serious than most of the other varieties because the rectal orifice into the posterior vaginal wall is usually of sufficient size to permit adequate emptying of the bowel, or if it is not large enough it may be easily found and readily enlarged by stretching or incision. Hence, life-threatening obstruction is rare. It does occur, however, when the communication is very small and high in the vaginal vault. Such cases may require colostomy to save the child's life, and are apt to be associated with double uterus. The writer has seen one such case. Much more commonly the rectum opens into

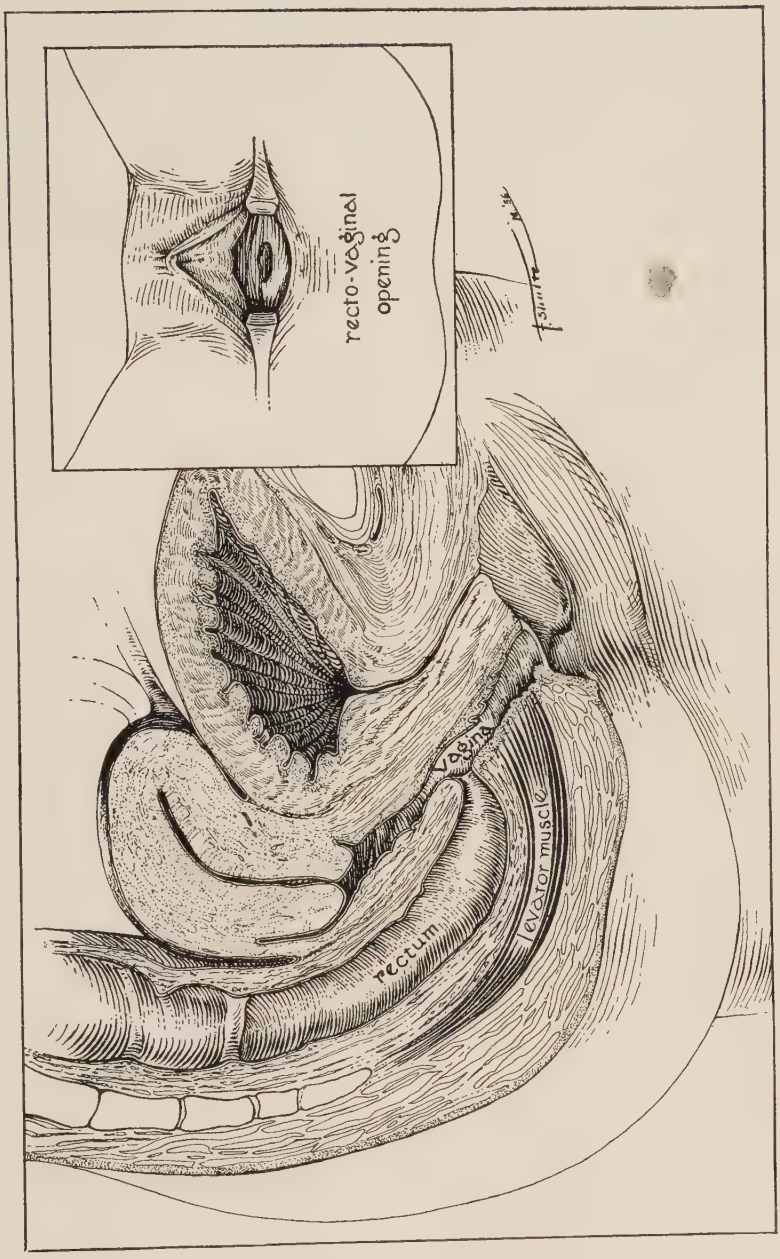

FIG. 1.—Sagittal section of imperforate anus and rectovaginal cloaca. Insert shows opening of rectum into vagina.

the posterior vaginal wall lower down, and indeed it has been said that the commonest location of the opening is in the fourchette.

So far as symptoms are concerned they are the obvious result of the abnormal location of the rectal orifice. Absence of the anus or a rudimentary dimple-like depression is the external evidence of the condition, combined with escape of meconium or stool from the vaginal outlet. There is usually an accompanying incontinence although sometimes there seems to be a partially effective sphincter-like power in the tissues about the rectovaginal opening. In social groups that are careless or unobservant, it is said that the condition may even escape detection and there are reports of women who had reached adult life, and had even borne children, without themselves being aware of their abnormal condition. As a rule, however, these patients suffer the disagreeable results of fecal incontinence and not infrequently the partial obstruction that goes with an inadequate rectal orifice. They have soiling, skin irritation, constipation, straining, and may develop one form of giant colon. Such a state of affairs clearly calls for efforts at cure or improvement.

So far as the writer has been able to discover, in a fairly extensive, but by no means exhaustive, search of the literature, there is only one form of operation described and utilized for the treatment of these lesions—that of Rizzoli.[2] In this procedure, an incision is made backward in the midline through the posterior vaginal wall, the fourchette, perineum and skin, from the edge of the rectovaginal fistula to the position that the anus would normally occupy. The rectum is detached from the vagina and drawn backward through the cleft perineum to its new position where it is sutured to the skin. The divided perineum and posterior vaginal wall are then sutured together in front of it, in order to reconstruct the normal relations. The results of this type of operation are generally described as good, but in some cases the repaired perineum has not held well and control has been unsatisfactory.

The writer offers a somewhat different technical attack with the same objectives—closure of the fistula into the vagina, restoration of the anus to its proper location and restoration of sphincteric control.

Operative Technic.—With retractors in the vaginal outlet to expose the rectovaginal fistula, a circular incision is made about this opening, separating the rectal and vaginal mucous membranes from each other. This incision is deepened about the rectal wall and the dissection is carried upward about the rectum on all sides until it is freely mobilized. As the anterior rectal and posterior vaginal walls often are in very close contact, meticulous care in dissection is sometimes needed to avoid making a hole in either viscus. The mobilization should be carried as high as possible without opening the peritoneum of the cul-de-sac of Douglas, which is to be avoided if possible. After this dissection is completed, the rectum lies free and separated in the space between the vagina in front and the sacrum behind, while below it the levator ani muscle forms a continuous layer across the perineal floor. The next step is to make a small oval removal of skin in the position where the anus should be. Sometimes this will be indicated by a dimple, or the sphincter

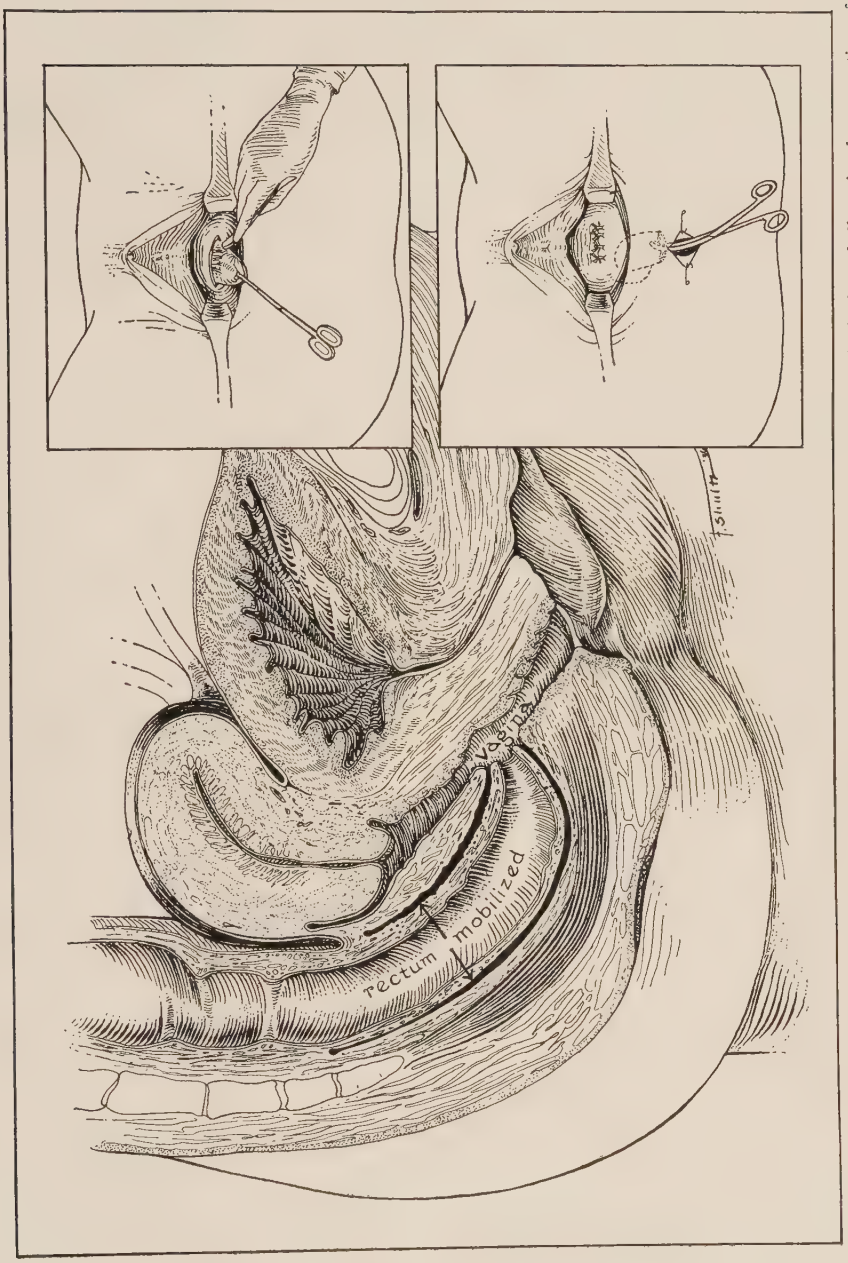

FIG. 2.—Mobilization of rectum by dissection from surrounding structures. Upper insert shows beginning of dissection by separation of rectovaginal orifice. Lower insert shows drawing of mobilized rectum downward through perineum to oval wound at normal anal location. The vaginal opening is shown here closed by stitches.

may be palpable under the skin. Often neither of these aids is present and the operator selects the anal location without special guidance. Through the skin wound that is to form the new anus a closed straight hemostat is thrust and worked, by blunt dissection, upward through the fascia and muscle of the levator until it penetrates into the free dissected space about the rectum. The passage through which the hemostat has made its way is stretched and enlarged carefully, without division of muscle fibers, until the index finger can be passed through it. Through this passage a clamp is then introduced upward from the perineum, until one can grasp the free end of the mobilized rectum, which is drawn downward and outward to the skin surface of the new anus. To do this properly without strain or tension, the rectum must first have been thoroughly mobilized high up, and the opening through the levator stretched enough to allow the gut to pass without difficulty. The rectum is now anchored in its new position with four quadrant submucous-subcutaneous sutures of catgut, and the free end of the gut sutured to the skin of the perineum with interrupted sutures of fine silk. By this procedure the perineal body and such muscle as exists in the pelvic floor have been preserved intact. The levator closes snugly around the rectum that has been pulled through it, and while the hole in the posterior vaginal wall is still open, a few catgut sutures may be taken in the levator and fascial structures in front of the rectum in its new position, to further build up a perineal body and rectovaginal septum. Lastly, the hole in the posterior vaginal wall is closed.

This method of operative treatment has been employed in the three cases reported herewith. It has yielded uniformly good results. The vaginal closure has been firm, without a leak. The perineal body is substantial. The rectum opens in its normal position. The grasp of the levator on the rectum as it surrounds it has afforded excellent control for both gas and feces. A few comments may be made on certain points affecting the ease and success of the operation. It is best postponed until the age of puberty is approached, as the structures then are larger and much more satisfactorily handled than in very young infants. As was previously mentioned, this is possible as there is usually sufficient egress of bowel content to avoid obstructive symptoms in this particular form of anomaly, although sometimes a colostomy may be necessary. When the selected age is reached, a few days' preliminary treatment in the hospital before operation, to empty the bowel completely, is very desirable. After operation, defecation is avoided if possible for seven to ten days, to permit the wound to solidify before subjecting it to the passage of feces. The daily administration of mild opiates effects this. After defecation has begun, warm sitz baths, twice daily, aid in the cleansing, healing and comfort of the wound. Before final dismissal, several digital examinations and gentle dilatations of the new anus and rectum should be carried out, but not earlier than the 14th day, in order that the newly healing wounds may not be damaged. A late survey of the cases, some months after opera-

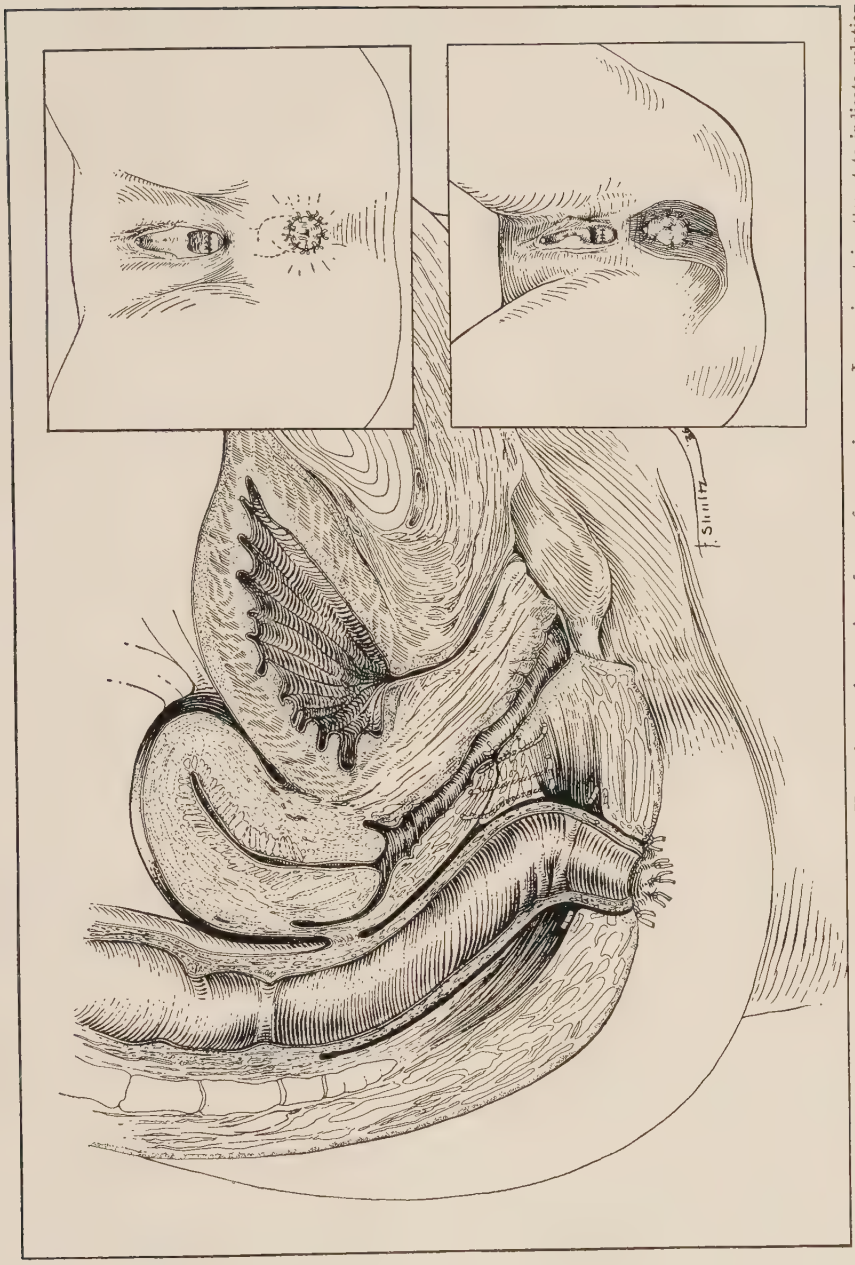

FIG. 3.—Sagittal diagram of completed operation. Upper insert shows view of same from perineum. Lower insert is attempt to indicate relation of levator muscle to new position of rectum.

tion, to determine the result, and to correct any tendency to stenosis, is desirable.

CASE REPORTS

Case 1.—No. 32812, Union Memorial Hospital. L. B., white girl, aged 13. Examination shows imperforate anus with a small scar, said to be due to an attempt to open the bowel by operation shortly after birth. When this failed, a colostomy was effected in the sigmoid, which is still open and functioning. In the posterior vaginal wall, one and one-half inches above the hymen, is a small opening marking the abnormal rectal orifice. The child is otherwise well developed, rather large for her age, and quite normal physically and mentally. The operation was performed as described June 27, 1934. On July 10, 1934, a digital dilatation was done. Convalescence was uneventful except for an intercurrent attack of pyelitis, and the patient was discharged after three weeks. She returned to the hospital one year later for closure of the colostomy, which was effected June 26, 1935, successfully and without incident. Examination at this time showed an excellent result. Normal appearing anus, good perineum, and excellent control of bowels without soiling.

Case 2.—No. 33117, Union Memorial Hospital. G. J. B., white girl, aged 13. Examination showed a small dimple where the anus should have been. The rectum opened into the posterior vaginal wall one inch above the hymen by an orifice, which had always been large enough to permit of easy defecation since birth. The child was otherwise normal, mentally and physically, well developed, and free from complaints, except for incontinence and irritation and excoriation about the vulva. The operation described was performed July 25, 1934. Wounds healed well, convalescence was uneventful, and the patient was discharged in three weeks. Examination some months later showed practically complete restitution of rectum, anus, perineum and vagina to normal with complete rectal continence.

Case 3.—No. 66891, Johns Hopkins Hospital. M. C., white girl, aged 10. Child was born with imperforate anus, the rectum opening through the posterior vaginal wall, low down. There were several other anomalies—an extra thumb on each hand, which was removed in early infancy without impairing the usefulness of hands, absence of coccyx, unilateral asymmetry of sacrum, fusion of several vertebrae, *etc.* For the past year or two, the child has had curious monthly attacks of mental disturbance, evidenced by confusion, irrational speech, and tendency to lapse into coma. These last several hours, and afterward she has no recollection of them. During the early years of her life, three or four attempts were made to correct the cloaca surgically, which succeeded only in bringing downward the inferior margin of the rectovaginal orifice so that it now lies practically in the fourchette. There is still incontinence except for solid stools, and vulval and perineal soiling and irritation. No sphincter muscle could be felt under the skin and there was no dimple to indicate the position of the absent anus. The operation described was performed December 30, 1935. During the first week there was some inflammation about the stitches closing the vaginal wound, but this subsided without breaking down of the repair, and the rectal suture held also. The child was discharged in three weeks, all wounds practically healed, the rectal orifice and lumen adequate and with practically normal control.

Summary and Conclusions

Rectovaginal cloaca of congenital origin with imperforate anus is a well known but rare anomaly. It is associated with incontinence of feces and gas in many instances, with the distressing results of such incontinence, but rarely with high grade obstruction that calls for surgical relief in the early hours of life. It is amenable to very satisfactory surgical correction, and a

method for such correction is herewith described, with a report of three successful cases. It is advisable to defer operative attack until the child approaches puberty, when the anatomic structures are easier to deal with than in the years of infancy.

REFERENCES

[1] Bodenhamer, Wm.: Congenital Malformations of Rectum and Anus. Samuel S. and Wm. Wood, N. Y., 1860.

[2] Rizzoli, Francesco: Memorie dell' Accademia delle Scienze dell' Institute di Bologna, 1857. *Idem*, 297, 1874.

Discussion.—Dr. William E. Lower (Cleveland, Ohio.)—I should like to present a case which is somewhat similar to that reported by Doctor Stone, except that the fistula was between the rectum and the urinary bladder. The child was born with an imperforate anus. I established a colostomy to relieve the acute condition, and then made an artificial anus, closed the fistula between the rectum and the bladder, and then closed the colostomy. The child is now 12 years old; she has no vagina, is almost reaching the age of maturity, and the secondary sex characteristics are developing. The problem now is, what is the next step? Shall I sterilize by roentgen therapy or perform another operation removing the uterus and the ovaries? I am presenting this as a problem. If any one can tell me just what should be done, I should appreciate it very much.

Dr. Franz Torek (Montclair, N. J.).—I had a case, similar to the one Doctor Stone presented, upon whom I operated when she was six months old. The fecal soiling was so disgusting that the parents could not tolerate it any longer. The procedure was the same that Doctor Stone has described. The fistula in the back portion of the vagina was closed; the rectum was moved backward and implanted at a new site.

That was 24 years ago. I never heard anything more of the patient until four months ago. She had in the meantime become married and divorced. The reason for the divorce was because of the absence of the perineal muscles, which resulted in loss of sphincteric control over the vaginal introitus.

On examination I found that at the operation when she was six months old, I had paid no attention whatever to the restoration of the perineal muscles. I told her that could be remedied. The operation was performed, and the result is perfect. There is a good sphincteric control. It was also possible to repair the sphincter ani, the anterior portion of which was open. She has since tested the local muscular functions with satisfaction to both parties.

Dr. Vernon C. David (Chicago, Ill.).—I think Doctor Stone's operation is a very much better operation than that described by Rizzoli, Delket or de Kermisson, all of whom employed the longitudinal incision, because, as Ombédonne says in his text-book, the rectum in that type of operation tends to return to the abnormal place, so that after a few months or years the situation is very much as it was before operation. I have seen six of these patients, children or infants, with atresia and vaginitis, and have operated upon two of them according to the method described by Doctor Stone, with one or two additions to the technic. Before mentioning them I should like to say that I think it is very important to allow these children to go until they reach the age of five to seven, because in two of these six patients, the rectum and the vagina have spontaneously and naturally separated them-

selves from each other, so that a perineum has formed between the vaginal orifice and the rectum, and the bowel has carried with it the muscles that surrounded the abnormal opening.

In these six children, three have remained continent with the abnormal opening, and if one decides to do an operation to replace the end of the rectum into another situation, which would be considered the normal site, it is very important to be sure that the external sphincter is present, because if the bowel is transplanted into a new place and incontinence develops, the patient will be a great deal worse off than she would have been with the abnormal opening and continence, and I really believe that continence is present in a number of these children.

In transplanting the rectum according to Doctor Stone's technic, you will notice the mucosa is sutured to the skin, and that results in a situation that is commonly seen after opening an imperforate anus, namely, an extrophy of the mucosa results; mucus covers the skin, and it is like a poorly performed Whitehead operation. I think that can be avoided. At least we did avoid it in two patients of this group by making skin flaps from the sides of the new opening of the bowel and allowing them to invert in this new position of the rectum, so that as the end of the bowel tends to retract, as it always does, it pulls these loose flaps of skin in with it. They can be turned in from the side, so that an anal canal lined with skin results, and if enough skin is turned in, it is a very helpful way to prevent stricture, which is another bad result one can obtain from transplantation of these abnormal openings to a new site.

Dr. Otto C. Pickhardt (New York).—Doctor Stone's presentation has been interesting and instructive. I should imagine that in a good percentage of cases the procedure outlined would be sufficient. However, each one of these cases presents certain individual difficulties which have to be overcome and where there is a question of a lack of length of the rectum, as in the appended case, which I would like to report, I think a more formidable procedure frequently has to be attempted.

Case Report.—A girl of seven was operated upon in 1928. The cloacal opening represented in Figure 1 is not quite correct. It was really situated well back in the

FIG. 1

posterior portion of the vagina. In this case also there was a dimple, but an entirely imperforate anus. The scar is the result of some previous operation (Fig. 1).

It was felt that because of the distance between the cloacal opening and the anal opening, that the simple operation of mobilizing the rectum would not be sufficient, and that a modified Kraske operation would help. Therefore, the usual incision was made, also one around the anal dimple, taking care to leave a bridge of tissue in this particular situation (Fig. 2).

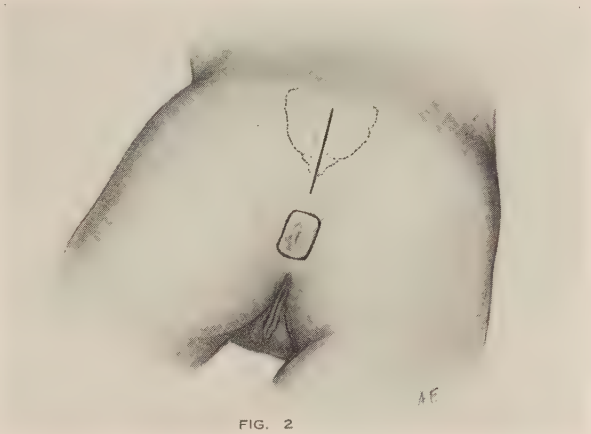

FIG. 2

With the coccyx and a small portion of the sacrum removed, it was an easy matter to mobilize the rectum and to obtain any desired length that seemed necessary at the time. The skin was removed over the anal dimple (Fig. 3). I think it is rather interesting that, in certainly all the cases that I have seen, and in most of those which have been reported, both the internal and external sphincter were almost invariably present.

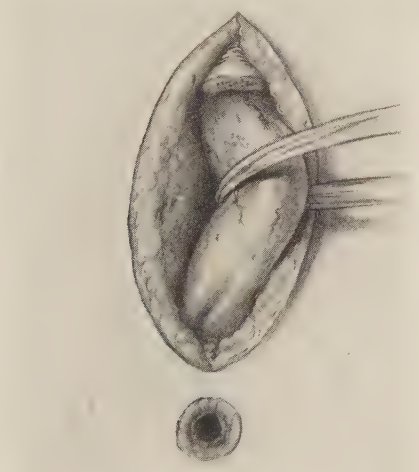

FIG. 3

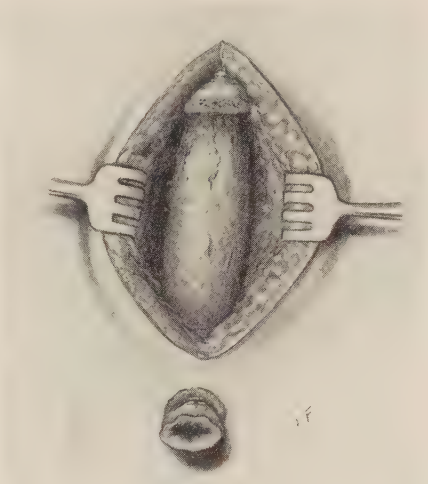

FIG. 4

After complete mobilization the rectum was brought out through the anal opening (Fig. 4). It is wise to leave a large cuff of mucous membrane in order to allow for the retraction that is sure to follow.

The various incisions were then closed, and in this particular instance the opening

in the vagina was so large that the sutures posteriorly could not be placed. The whole vaginal wall, therefore, was brought forward and sutured to the fourchette (Fig. 5).

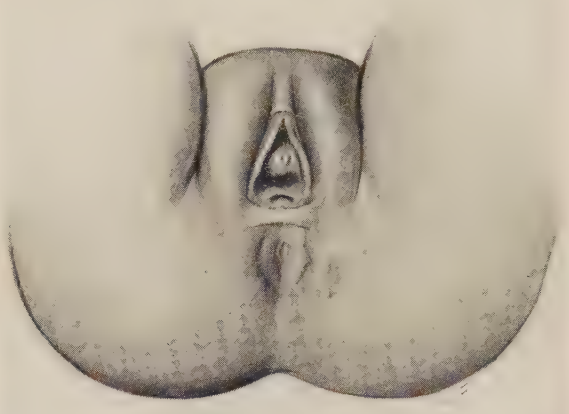

FIG. 5

Another point of interest is the time when the first bowel movement should take place. I waited 13 days in this particular instance. The wounds healed kindly, without infection, and at the end of 30 days the patient had very definite sphincteric control. For the first two or three years, regularly, every month, bougies were passed. At present the child is in perfectly normal condition.

Dr. Harvey B. Stone (Baltimore, Md.) closing.—I think Doctor Pickhardt is right when he says there are much more complicated cases than those I showed, that require extensive and elaborate operations. As a matter of fact I think some of them are probably insoluble.

Such a case, which I referred to briefly, was a patient who had a double uterus, with a very small tubular end of the rectum opening into the very apex of the vaginal vault, between the two cervices, with no gut below that point at all. I felt that case could not be corrected by any measures that I knew of, but I am sure that some less difficult ones might be corrected by the approach Doctor Pickhardt has described.

I have no answer to Doctor Lower's problem. It would take a wiser man than I to tell him what to do in such a condition. I think Doctor Torek's case emphasizes the advisability of deferring operation in these children until they are out of the infant class, certainly until they are four or five years old, and I believe, better, until they are approaching puberty.

I appreciate Doctor David's suggestion for handling the suture line at the margin of the skin-mucosa anastomosis. I am quite sure that is a valuable addition, and shall employ it at the first opportunity.

OPERATIVE INSULIN CRISIS IN RESECTION OF THE PANCREAS

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SURGERY of the pancreas has come to the fore during recent years because of the more general recognition of cases of hyperinsulinism. So far, the reports of about 35 such cases coming to operation have appeared in the surgical literature. Because of its rarity each case has taught us something new. The instance which we have encountered is unique in some of its aspects, and teaches a lesson well worth remembering.

The attitude of most surgeons writing on the subject has been that resection of the pancreas for hyperinsulinism is a relatively benign procedure since no fatalities have been recorded. We had the unfortunate experience of operating upon and reporting the first case to die from resection of the pancreas. The cause of death and suggestions of means for preventing similar occurrences in the future form the theme of this report.

Case Report.—W. Y., a Negro male, approximately aged 45, was admitted to Waverly Hospital May 7, 1935. Fifteen years ago he was struck on the head with a blunt instrument and received a laceration on the forehead about three inches long. The skull was exposed but not thought to be fractured. He remained unconscious for about two days, but after three weeks was able to resume work and was perfectly normal, mentally and physically, until the beginning of his present illness three years ago.

Present Illness.—Three years ago he developed a generalized convulsion accompanied by foaming at the mouth, and passed shortly into a stupor from which he could not be aroused. He remained in a semistupor for about two days. Sweating was profuse, and he was cold and clammy during this attack. During the past three years he has had these seizures with the same general pattern at intervals varying from two weeks to two months. His wife could always predict the onset of a convulsion by his confused speech, clamminess of the skin, and beads of perspiration on his forehead. Various mental reactions occurred from placidity to throwing of chairs, attempting to climb walls of room, pulling off clothes, and delusions of persecution. Recovery from an attack would usually take a day or more. His wife claimed that the attacks seemed to occur on days when he had not eaten much, and that she had never seen him have an attack when he was eating normally.

Examination was negative including superficial and deep reflexes, except for a slight scar over the left eyebrow, blood pressure 140/90, and poor oral hygiene. Spinal fluid and blood Wassermann reactions were reported negative.

Course and Laboratory Data.—The diagnosis was not suspected until the fifth day of his stay in the hospital at which time he had a rather typical convulsion with its characteristic prodromal symptoms. Blood for sugar determination was taken at this time, and he was given 35 cc. of a 50 per cent glucose solution intravenously. In about ten minutes he had recovered his normal mental status. The blood was reported to contain 75 mg. glucose per 100 cc. The next day he was found to be cold and clammy; another blood sugar was taken. This was reported to contain 55 mg. After his usual dinner his symptoms disappeared. The following day blood sugar readings were taken before, and a half-hour after, each meal with the following results: 8:00 A.M. (fasting)

—57 mg.; 8:30 A.M.—115 mg.; 12:00 NOON—111 mg.; 12:30 P.M.—105 mg.; 6:00 P.M. —95 mg.; 6:30 P.M.—80 mg. Next day, at the suggestion of Wilder, food was omitted until 5:00 P.M. Sugar at 7:30 A.M. was 50 mg.; at 11:30 A.M.—55 mg.; at 4:30 P.M. —55 mg. No convulsions or untoward symptoms appeared during this period of starvation. The next day a glucose tolerance test was run. *Before the ingestion of 100 Gm. of glucose the blood sugar was 30 mg.; at one-half hour it was 111 mg.; at one hour, 80 mg.; at two hours, 80 mg.; after three hours it had dropped back to the original 30 mg.*

During the next few days the patient was given his regular diet with the addition of intermediate feedings of low carbohydrate vegetables. One chemistry taken at midday during this period showed 36 mg. of sugar. After this disappointing response to diet we began to test his response to drugs. He was given 1 cc. surgical pituitrin, before which the sugar was 36 mg. Fifteen minutes after the administration of pituitrin it had risen to 57 mg. In 30 minutes it had dropped back to 50 mg. Adrenalin was tried the following day. Before administering 10 minims of adrenalin it was 56 mg.; 15 minutes later it was 60 mg.; at the end of 30 minutes it was 50 mg. Atropine was next tried, 1/100 gr. Before, it was 47 mg.; 15 minutes after, it was too low to read (below 20 mg.); a half-hour afterward, it was still too low to read. Because of the rather startling drop, this drug was tried again next day with the following results: Before the atropine, 47 mg.; 15 minutes after, 40 mg.; 45 minutes after, too low to read; one hour after, too low to read. He was tested with physostigmine, the pharmacologic antagonist of atropine. Sugar reading before this drug was 50 mg.; 15 minutes after, 1/100 gr. physogtigmine, it was 50 mg.; 45 minutes after, it was 55 mg.; and one hour after, it was 60 mg. It will be noted that this rise is a little higher and longer sustained than that after adrenalin or pituitrin.

Judd assumed that a disturbed vagal control of the pancreas was responsible for its peculiar behavior in spontaneous hyperinsulinism when no tumor was found. We believe that our experiment with physogtigmine and atropine raises his assumption to the level of a reasonable assertion.

After this thorough trial with diet and drugs had failed to maintain the blood sugar at a reasonably safe level, and, bearing in mind the possibility that an adenoma or carcinoma of the pancreas might exist, we considered an exploration definitely justified. The operative procedure will be described in some detail to emphasize the fact that it was rather benign *per se* and easy of accomplishment.

Operative Procedure.—After the usual preoperative preparation plus a cup of very syrupy coffee about one half-hour before going to the operating room, he was anesthetized with ether. An upper midline incision, from the ensiform to the umbilicus, was made. The stomach and transverse colon were delivered and a window made in the gastrocolic omentum. The stomach and transverse colon were retracted and a very satisfactory exposure of the pancreas obtained. The anterior surface of the gland was carefully inspected for adenomata, but none was found. The decision was then made that a resection offered the patient his only chance of relief. This we proceeded to do.

The peritoneum was incised near the tail of the organ, and this part gradually freed. As the process of freeing went forward, an anomalous splenic vein with a large, separate pancreatic branch was discovered. This large pancreatic vein was ligated about halfway up the body of the pancreas and further freeing of the body of the organ accomplished without difficulty. Bleeding was minimal. A rubber covered, right angle clamp was placed across the pancreas about midway of the body, and a V-shaped incision made to remove the freed portion. Lock sutures were inserted in the cut surface for hemostasis, and the clamp removed. Two large cigarette drains were placed in the bed of the resected portion and brought through the left part of the rent in the gastrocolic omentum. The cut edges of the omentum were approximated with small catgut ligatures. The closure of the peritoneum was begun and halfway completed when the patient stopped breathing. Artificial respiration was begun, 7½ gr. of caffein sodium benzoate were given intravenously, and 15 minims of adrenalin were injected directly

into the heart. In about two minutes the heart ceased to beat, and, although it was massaged through the diaphragm, the rhythm was never reinstituted. The patient's death was recorded as an anesthetic one until the interesting observations appended were considered.

During the course of the operation 1,000 cc. of a 10 per cent solution of glucose had been given intravenously. This was begun about five minutes after anesthesia had been effected, and was completed in about 20 minutes. Blood for sugar determination had been taken, first, after surgical anesthesia was attained; a second specimen, after the pancreas had been exposed; and a third, after the resection had been completed. They were reported to be 46 mg., 30 mg., and "too low to read," respectively.

Discussion.—This gradual drop to such a level during the operation of resection has not been recorded by other observers, *and is the interesting feature in this case.* Other investigators claim that ether is the anesthetic of choice because it tends to raise the blood sugar. This anesthetic agent, along with 1,000 cc. of 10 per cent glucose intravenously during the course of resection, is generally thought to be positive insurance against the lowering of the blood sugar. The fact that the blood sugar readings in our case became progressively lower would definitely discredit this claim, or certainly prove that it is not universally true. This progressive lowering to such a level made us believe that the cause of death in this patient was a hypoglycemia which, when coupled with the shock attendant to his operation, was incompatible with life and was the immediate or direct cause of his death. It has also led us to suggest that there is an entity, heretofore unrecorded, which we have chosen to call "operative insulin crisis." We suggest that it is due to manipulation of the gland and liberation of an overwhelming dose of insulin into the blood. We do not believe such a lowering of the blood sugar would have occurred independent of this operative procedure in our case while 100 Gm. of glucose were placed at the disposal of his pancreas. If our supposition is correct—that such an entity exists—and we believe that the findings in our case are proof of such a premise, this entity must be reckoned with in future resections of the pancreas. It may be that larger amounts of glucose during the operation will ward off such a crisis. We believe that this entity is a very definite threat against the safety of any patient undergoing similar resection, and that more consideration should be given to the administration of glucose in adequate amounts.

The published reports of end-results from resection of the pancreas have not been encouraging. Isolated reports of good results from radiation of the pancreas have appeared in the recent literature. The latter fact, together with our disagreeable experience with resection and the poor end-results of other observers, has led us to offer the warning that all conservative measures, including roentgenotherapy, should be tried, and that further search should be made for other non-operative measures for the relief of those patients *in whom no tumor is found.* The use of physostigmine, which we report, by inhibiting the function of the pancreas through its vagal control may offer some relief to borderline cases. We believe that further trial of this drug is warranted. Certainly, after a review of the end-results,

we must contend that any other non-operative measures suggested will be more than welcome.

CONCLUSIONS

(1) A case of spontaneous hyperinsulinism, in which death resulted from resection of the pancreas, is presented.

(2) A condition, which we have called "operative insulin crisis," is presented and discussed.

(3) An experiment tending to prove the disturbance in vagal control of the pancreas in hyperinsulinism is described and discussed.

(4) The suggestion is made that the attitude toward resection of the pancreas be tempered with a little more caution and restraint in the future, and that more stringent measures be employed to maintain a safe level of the blood sugar during operation, when it is of necessity performed.

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DISCUSSION.—DR. ALLEN O. WHIPPLE (New York, N. Y.).—It would seem to me that this very interesting case report of Doctor Guerry's is entirely different from the other cases of pancreatectomy that have been reported in the literature. I regret that Doctor Graham is not here to report on his remarkable case and brilliant cure following a partial resection of the pancreas in an infant for hypoglycemia. I know of two other cases, that have not as yet appeared in the literature, that have been entirely relieved of the hyperinsulinism following partial pancreatectomy in which an adenoma was not found. I operated upon a similar case a month ago in a woman of 32 who gave all of the classic findings of hypoglycemia, and who had failed to respond to all forms of dietetic and medical regimens. She was referred to me from the Neurologic Institute of Columbia University. In the course of a very careful exploratory search I was unable to find an adenoma, and therefore resected four-fifths of the pancreas, leaving a small portion of the head. In this patient the rise in blood sugar values was more gradual than I had expected; but in the period of a few weeks the readings have risen from 50 to 75 and the patient has been entirely relieved of her attacks of unconsciousness and hypoglycemia.

In the absence of finding an adenoma in the pancreas itself, a careful search should be made in adjacent organs for an heterotopic pancreatic tissue, particularly in the region of the duodenum, and for a possible Meckel's diverticulum containing pancreatic tissue.

I should like to call attention again to the great advantage of the transverse incision over the vertical incision in these operations for hypoglycemia; and to emphasize again the great superiority of silk technic in operations upon the pancreas.

DR. ROSCOE R. GRAHAM (Toronto, Ontario).—Doctor Gallie and I have had an opportunity to study, in conjunction with our medical confreres, a patient with a history similar to the one Doctor Guerry has presented. We felt we were justified in advising exploration, but at operation found no tumor. This was the first case we have had an opportunity of studying in which no tumor was present. We decided that she should have a resection of the pancreas. In view of the disappointing results which have been reported following such a procedure, we felt we should be very radical, and believed we had left but a very small portion at the end of the operation.

Our estimation of the bulk of pancreas left would be equal to a mass not greater than the distal phalanx of the thumb. We were very much chagrined to find that the weight of pancreas removed, despite what we considered a radical resection, was only 20 Gm. We believed, however, that this must originally have been a very small pancreas.

Our patient, like Doctor Whipple's, did not have an immediate fall in blood sugar. However, at the end of ten days the blood sugar was stabilized at a normal level, and we believed our efforts were likely to be rewarded with relief.

Following the administration of a fairly generous and normal diet, she carried on until the end of the fifth week, when she was again seized with convulsions. It is now about five or six months since her operation, and as far as we can see she is not in the least improved, as compared with her preoperative state.

Professor C. H. Best, who has charge of our Department of Physiology, was good enough to assay the removed pancreatic tissue, and to determine the number of insulin units present. The highest incidence of insulin units in pancreatic tissue which they had previously encountered was 2,500 to 3,500 units per kilogram of tissue. The pancreatic tissue which we had removed assayed 8,000 units per kilogram, which they considered incredibly high.

We are now confronted with a problem more difficult than the original one. I should like to have an expression of opinion from Doctor Guerry as to whether he thinks the removal of a greater amount of pancreatic tissue would have solved our problem. Doctor Whipple's suggestion that we may have missed some aberrant pancreatic tissue may be true; in any event, we failed to recognize it as such.

Dr. Le Grand Guerry (Columbia, S. C.) closing.—We wished to add this little bit of evidence, in order to accumulate, on this subject, such a body of facts and statistics as to give us some certain, sure path in which to walk. Our experience in operative hyperinsulinism is confined to this one case, and the theme of this paper is the fact that our case presented a feature not yet recorded in the literature on hyperinsulinism. Here is a patient with definite hyperinsulinism, given a large dose of sugar by mouth before operation and after the anesthetic was begun, he was given 1,000 cc. of 10 per cent glucose intravenously, and every one of the blood sugar readings taken during the operation, *i.e.*, after surgical anesthesia had been induced, after the pancreas had been exposed, and after it had been resected, showed a progressive fall and the last reading was "too low to read" (below 20 by the standard used). We are holding this up as a point to be reckoned with in the future handling of this condition and are suggesting that there may be such an entity as "insulin crisis," which may occur in any operative procedure involving extensive manipulation of the pancreas and the liberation of large amounts of insulin into the blood stream.

BENIGN CICATRICIAL STRICTURES OF THE BILE DUCTS

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STRICTURES of the hepatic and common ducts are due to a variety of causes which need not now be enumerated. At present, the end-results of operations for benign strictures, due to cicatricial tissue in the wall of the duct, will be considered. Of this type of stricture partial or complete division of the common or hepatic duct in the course of cholecystectomy is unquestionably the most frequent cause. This unfortunate accident, not always due to ignorance or inexperience, may happen to the most skilled surgeon. Abnormalities in the course, length and termination of the cystic duct, and variations in the course, origin and distribution of the cystic artery, fully described by different writers, are important predisposing causes. The pressure of an hemostat on a portion of duct wall, occurring in the attempt to secure a bleeding cystic artery, is also a competent producing cause of subsequent stricture—usually in the hepatic duct just above its junction with the cystic duct. Benign strictures are usually associated with biliary fistulae. When the stricture is acute and complete, as after accidental ligation of the hepatic duct during cholecystectomy, jaundice develops rapidly, symptoms of cholemia and liver incompetency appear, and a fatal issue is averted only by the loosening of the ligature and the formation of a biliary fistula. On the other hand in strictures which form slowly, symptoms of obstructive jaundice are intermittent, appearing when the associated fistula is temporarily occluded. As the stricture tightens after a period of months or years, obstructive jaundice becomes persistent and the patient eventually succumbs to the resulting liver cirrhosis.

As long as the flow of bile through the fistulous tract is unimpeded, the total exlusion of bile from the intestine usually causes disturbances of digestion, the loss of flesh and strength and ultimately a condition of osteoporosis. Exceptionally such exclusion may continue for months or years without impairment of the general health. In the case reported by Moschowitz no bile passed into the intestine for three years; in another case reported by the writer, lasting 100 days, the sole complaint by the patient was the inconvenience of the biliary discharge. In both patients the bile suddenly found its way into the intestine, the biliary fistula remaining permanently closed. This unexpectedly fortunate result was probably due—not to the restoration of the normal duct channel—but to the formation of an adventitious opening in the adjacent intestinal wall by infected bile in much the same way as in the pointing of an abscess.

Symptoms of benign stricture in the absence of previous operation are usually those of the gradual development of jaundice with or without occa-

sional attacks of cholangeitis. When pain is present, an impacted calculus in the common duct is the probable preoperative diagnosis. When pain is absent, especially in patients over 40, malignancy in the head of the pancreas is suspected.

Benign strictures vary in their location and extent. Strictures of the hepatic duct just above its junction with the cystic duct, as already mentioned, usually follow cholecystectomy and are localized. Strictures in the common duct frequently due to the cicatricial contraction of pressure ulcers from impacted calculi and limited in extent, are more common at or near the ampulla. Strictures due to septic cholangeitis are generally diffuse and may involve the greater part of both common and hepatic ducts.

Operative measures which vary according to the location and extent of the stricture may be arranged in the following groups:

(1) End-to-end anastomosis after excision of the stricture when the orifices of the duct can be approximated without undue tension.

(2) Choledochoduodenostomy when the stricture involves the terminal portion of the common duct.

(3) Hepaticoduodenostomy, -gastrostomy, or -jejunostomy when the stricture involves such a large portion of the common duct that either of the preceding operations is impossible.

(4) Reconstruction of a new duct by the tube method (Wilms).[116]

(5) Implantation of the biliary fistula into stomach, duodenum or intestine.

(6) Cholecysto-enterostomy in strictures of the common duct when both the gallbladder and cystic duct are normal.

(7) Dilatation of a stricture with the insertion of a buried tube.

(8) Choledochotomy or simple division of stricture.

(9) Hepato-enterostomy, *i.e.*, the approximation of denuded liver tissue to duodenum or small intestine where the stricture involves the hepatic duct within the liver, in which dilatation of the stricture cannot be carried out or has failed to give relief.

While striking results at times have followed each one of these measures with the exception of the last two, failures are not uncommon. Either the stricture recurs within a year or earlier, or a septic cholangeitis, of increasing intensity eventually proves fatal. Recurrence of the stricture is less likely if, in the operative anastomosis, the mucous membrane of the divided ends of the duct or of the duct and intestine can be approximated and sutured without tension. The absence of infection then favors primary union, and, if the line of suture is not torn apart by the later withdrawal of the tube from within the duct, stricture ought not to recur.

If the ends of the divided duct or of the duct and intestine cannot be properly approximated, the resulting gap may be bridged by the insertion of a rubber tube which is then invested by omentum. There is some evidence to show that the granulation tissue encircling the tube becomes lined by the extension from above and below of the normal epithelium of the duct and if, in

the case of obliteration of the common duct, the tube extends from the hepatic stump into the duodenum, by the extension of the duodenal epithelium upward as well. The capacity of cells lining mucous membranes to proliferate and to extend over adjacent granulating surfaces has long been unquestioned. Doctor Rohdenburg of the Lenox Hill Hospital told the writer that, ten years after the establishment of a gastric fistula for a stricture of the esophagus, the excised fistulous tract differed from the mucous membrane of the stomach only in that the submucous glands were more "stumpy" in character. He also called attention to the fact that, in complete fistula in ano, the fistulous tract becomes lined by an extension of the rectal mucous membrane from within outward and by an extension of the epidermal cells from without inward. Phemister[86] of Chicago has also demonstrated the extension of epidermal cells over the granulating surface of an osteomyelitic cavity in the lower end of the femur. Such cellular proliferation is shared by the lining epithelium of the hepatic and common ducts and the duodenum. Assuming that a segment of a bile duct can be reconstructed in this way, such a reconstructed duct ought not to undergo any marked diminution of its lumen through the contraction of the organized granulation tissue of which it is in large part composed. That such extension does not always occur, however, has been shown by Colp,[16] who bridged an interval between the divided ends of the common duct with a rubber tube invested by two layers of connective tissue of the gastrohepatic omentum. Sixteen months later the patient died of myocarditis without return of any symptom of biliary obstruction. On autopsy the reconstructed segment of duct was found to form a canal epithelially lined but smooth walled, and without the usual duct structure. There was no stenosis or narrowing. On microscopic examination the reconstructed segment consisted of dense laminated connective tissue covered by one layer of flat spindle cells. The absence of any stenosis in this case is of the greatest interest. On autopsy in Freeman's case, the reconstructed part of the duct was patulous although small, about the size of a "broom straw," but fluid could easily be injected through it.

Similarly, the wall of a biliary fistula is supposed to become lined with epithelium by extension of the mucous membrane of the duct and by the duodenum as well, if it is connected with that structure. In Jenckel's case the external orifice of the fistula became lined with duodenal epithelium within 11 days. He also emphasizes the capacity of the epithelium of the duct to grow and thinks that this process is hastened by its dilatation. Unfortunately in his second case which succumbed 12 years after a reconstruction operation, the autopsy record failed to mention any microscopic examination of the reconstructed duct. It stated, however, that two centimeters below the transverse fissure of the liver a callous like mass was found in which no canal could be identified macroscopically. The hepatic tributaries were not dilated and he was of the opinion that the reconstructed duct functioned to the end, and that cirrhosis of the liver, together with amyloid degeneration of the kidneys and spleen, caused the patient's death.

Roeder[90] of Omaha has made a valuable contribution to this subject.

Before implanting a biliary fistula of four months' duration into the duodenum, the terminal 1½ cm. of this fistulous tract were excised; examination showed patches of columnar epithelium and gland like structures resembling more the mucous membrane of the duodenum than that of the common duct. On the other hand, Cave reports no epithelial lining in the wall of a biliary fistula 106 days subsequent to its formation.

Recurrence of stricture may be due to errors in the use of the rubber drain. Thus, after end-to-end suture over a T tube emerging through the line of suture, stricture may follow if, in its withdrawal, the ends of the duct, already advanced in union, are torn apart. To diminish the risk of this accident the writer suggested the use of collapsible or soft rubber, around which a strong piece of silk, firmly tied, was brought out of the abdominal incision. In two cases treated in this manner the withdrawal of the tube was evidently harmless as all discharge of bile ceased within 48 hours. In suture of a duct, incompletely divided, the tearing apart of the line of suture is less likely to take place. The writer also suggested the bringing out of the tube through a separate opening in the wall of the duct either above or below the line of suture. This procedure has been practiced by a number of surgeons including Horgan[48] of Washington, who advocates the use of a special L shaped tube. The divided duct is sutured over the inserted shorter limb of this drain, while its longer limb, making an oblique angle with the shorter limb, emerging at the separate orifice, is then brought out of the abdominal incision. Horgan reports three brilliant results in which this technic had been employed. Dr. S. W. McArthur,[76] in place of a T tube, uses an L shaped tube passing upward in the choledochus with a small rubber catheter passing through the same opening down into the duodenum. The latter can be utilized for irrigation and medication.

The use of a rubber tube so large as to unduly compress the mucous membrane of a duct, into which it has been inserted, must be avoided. Too stiff rubber tubes are also objectionable, as their withdrawal from the duct is liable to rupture a line of suture, or may damage the wall of a reconstructed duct, if that method of duct repair has been attempted. Furthermore, the extremity of such a tube, if it becomes disengaged, may produce pressure necrosis of the wall of a hollow viscus. Von Redwitz[100] (personal communication) related a case in which an ulcer of the stomach was caused in this manner, and Roeder a case (personal communication) in which the end of a 14 cm. catheter, over which a biliary fistula had been anastomosed to the duodenum, passed two months later, through the anastomosis or wall of the duct, into the peritoneal cavity, and about three months afterward penetrated the pelvic sigmoid, causing a fatal peritonitis.

In duct reconstruction with a buried tube, the retention of the tube as well as its removal when the duct is fully formed presents certain difficulties. It was at first hoped that the buried tube would remain permanently fixed in position or that it would ultimately disintegrate. It was found, however, that a tube so fixed usually became blocked in the course of a few months

or possibly years with precipitated biliary sediment which also coated its outer surface, causing recurrence of jaundice which was relieved only by the removal of the tube. Shenstone, of Toronto, advocates the monthly instillation of two or three drops of sulphuric ether to keep a permanent "T" tube free from precipitated sediment. In a case cited this has proved effectual over a period of two years. Cases of this accident have been reported not infrequently, including one instance (Judd[54]) in which such an incrusted tube remained in situ for six years. Premature discharge of the tube, predisposing to a recurrence of the stricture, may be prevented by the suture of its upper extremity to the wall of the hepatic stump, nonabsorbable material being used. L. L. McArthur[75] endeavored to secure the same result by forming a reverse cuff on the end of the tube which then ordinarily remained in situ for four or five weeks.

To prevent its premature discharge and to permit its removal at the will of the surgeon the following technic is suggested by Voelcker.[102] The buried tube attached to the hepatic stump above, and in the absence or obliteration of the choledochus, penetrating the duodenal wall (Witzel) below, is then brought out through a separate opening in the anterior duodenal wall (Witzel), and through the abdominal incision, to the edge of which it is firmly attached. This measure has been employed to a limited extent by surgeons abroad with variable results. The possibility of a duodenal fistula is obvious. If, as a result of the operation, the passage of duodenal contents is obstructed by its acute angulation, a posterior gastro-enterostomy is added.

One of the most frequent, and justly dreaded complications, common to all operations for the relief of benign stricture, is cholangeitis. This complication, if mild in character, occurs in occasional attacks of short duration during the first year or two after the operation and then ceases. It is probably due to temporary swelling of the duct at the point of suture. The more severe cases are due to infection, the source of which is still unsolved. The fact that it seems to occur with greater frequency the more remote the anastomosis is from the papilla, points to an intestinal origin. That it may arise within the biliary tract is also quite probable. Cholangeitis is unquestionably favored by any obstacle to the outflow of bile. Efforts to eliminate this unfortunate complication by making the anastomosis between the biliary and intestinal structures, in a loop of gut from which the normal intestinal current has been diverted, have not diminished its frequency. Irrigation of the duodenum both before and after operation with a solution of magnesium sulphate may be of some value. Douglas[18] has suggested the administration of large doses of bile salts and Williams the administration of ox gall in order to diminish the fetid character of acholic stools, and more recently the use of secretin to promote the flow of bile has been suggested by Prewitt.

Before proceeding to a review of the end-results of the different operations for the relief of benign stricture, it is important to emphasize that extensive adhesions between the parietal peritoneum, omentum, the edge of the liver, the pylorus, duodenum, and the hepatic flexure of the colon, obliterate anatomic landmarks and make the exposure, recognition, and orientation of

the stricture hazardous and difficult. Discretion may therefore be the better part of valor, particularly in recurrent strictures when biliary cirrhosis, and possibly infection, have impaired the function of the liver. Under such circumstances after biliary drainage has been established the actual reconstruction of the duct may be advantageously postponed to a later time when the jaundice has subsided and the function of the liver has, at least, been partially restored. The frequency with which strictures recur, each recurrence requiring further operation, is a common experience to all. Usually more than one and, at times, as many as five or six attempts have been made to relieve the patient in the course of two or three years. This multiplicity of operations at least testifies to the ability of patients with damaged biliary tracts to endure formidable operative procedures.

The writer has collected 38 cases of duct reconstruction by end-to-end anastomosis reported in surgical literature and from personal communications of colleagues in this country and abroad. While failures have not been infrequent, the number of successful end-results is most gratifying. No less than 14 patients have remained well from ten to 19 years and ten others from five to ten years. In each instance the patient, if living, was symptom free at the end of the period of observation. Three patients (Stetten,[95] Downes[19] and Eliot[22]) died from intercurrent disease at the end of 19, ten, and 18 years respectively. It is consequently not unreasonable to conclude that the relief may be considered permanent, if patients have remained free from recurrence for five years. Patients who have remained well for ten years or longer include those operated upon by Douglas[18] (two cases), Matthews,[65] Downes (two cases), Homans[47] (two cases), Riggs,[89] Schweizer,[94] Papin,[83] Horgan, Stetten, and Eliot[23] (two cases). Many of these, in whom the relief may be said to be permanent, suffered from mild attacks of cholangeitis for the first and second year after operation. In one of them—active and in good health at the end of 13 years (Downes)—these attacks persisted for three years after operation and, at the present time, indiscretion of diet is usually followed by a short "bilious" attack. In both cases reported by Douglas (well 11 and 14 years after operation) these attacks occurred, in one, not until the second year after operation. Similar experiences have been had by Matthews, Stetten, Seward Erdman,[27] and Eliot. In Erdman's case the attacks still continue, though with decreasing intensity, three and two-thirds years after operation.

Exceptionally attacks of cholangeitis have been so severe as to have proven fatal. A patient of McArthur's succumbed 18 months after operation and Fiolla's patient, after remaining free from cholangeitis for two years, died subsequent to a particularly vicious attack. In one of Judd's patients an abscess of the liver developed which was, however, successfully drained. The fact that mild attacks of cholangeitis frequently subside after a year or two justifies conservative treatment. Only when they increase in frequency and intensity with persistent and deepening jaundice is further operation indicated.

HEPATODUODENOSTOMY.—To the 41 cases here reported of hepatoduodenostomy, performed by various surgeons, must be added 15 reported by Wal-

ters[103] without detail, one being alive and well five years after the operation, and several others for shorter periods. Of the 41 cases, 11 remained well for from ten to 20 years. The fact that these patients were symptom free at the end of the period of observation justifies the conclusion that the cure may be considered permanent. As in cases treated by other methods, cholangeitis was a frequent postoperative complication. In the case reported by Leriche,[60] the attacks of cholangeitis which ultimately caused the patient's death did not become severe until two and one-half years after operation. Septic cholangeitis, terminating fatally from abscess of the liver, was also observed in Melchior's[69] patient.

Usually leaving a small tube in the anastomotic opening for a considerable period of time after operation, at least for several months, is advocated, and to its early passage recurrence of the stricture has been ascribed. J. F. Erdmann[25] removed a tube completely blocked with precipitated biliary material five and one-half months after operation, and Judd, in a patient who developed cholangeitis four years after operation, removed a calcified tube six years after the hepatoduodenostomy. This is by far the longest record found in which the tube has remained in situ. On the other hand, some surgeons advocate anastomosis without a tube or its early removal. Anastomosis, in which the respective mucous membranes can be approximated without undue tension, offers a favorable condition for this method of treatment. In Moritz Cohn's[15] case in which the patient was symptom free at the end of 12 years, the tube was removed on the twelfth day.

The first successful operation of hepatoduodenostomy was probably performed by W. J. Mayo[71] in 1904. Fifteen years later this patient was symptom free. It is a pleasure to call attention to the striking success of LeGrand Guerry,[41] who relieved seven consecutive patients without a death and, so far as is known, without symptoms of recurrence. This surgeon modestly expresses a preference for hepatoduodenostomy rather than for choledochoduodenostomy, as two patients, earlier operated upon by the latter procedure, succumbed to pneumonia and cholemia respectively. No other surgeon has enjoyed such freedom from mortality.

Choledochoduodenostomy.—Twenty-seven cases of this operation for stricture, gathered by the writer from the literature and personal communications, include a number in whom sufficient time has not elapsed to determine the final result. Two patients (Behrend[5] and Peterman[85]) were symptom free 19 and 12 years respectively after the operation. Two others (Finsterer and Guerry) were symptom free six years after operation. Caves'[14] case, in which the anastomosis was done with a Murphy button, was well nine years afterward. Several others were symptom free for shorter periods. The fact that stricture is more common in the hepatic duct, owing to the greater frequency of accidents during cholecystectomy, probably accounts for the relatively small number of cases of this type. The number is sufficient, however, to prove that, as in other operations for stricture, cholangeitis is a common, though perhaps a later and less severe, complication. In Downes' case, death did not occur until six years after the operation and an equal time elapsed

in the case reported by Sasse[91] in which, at the time of operation, a pneumococcus infection of the duct was present. In Judd's case an abscess of the liver was successfully drained, the patient being well three years afterward.

To determine the exact nature of the stricture—whether cicatricial or inflammatory—when situated at or near the ampulla, is much more difficult than in strictures in the more accessible portions of the hepatic or common ducts. In both conditions choledochoduodenostomy is justifiable. If the stricture is cicatricial, the anastomotic channel is permanent; if inflammatory, the new channel, providing adequate drainage as long as the stenosis continues, is probably gradually eliminated when the patency of the duct is restored. Such restoration is graphically demonstrated in an autopsy reported by Finsterer, the patient dying from cancer of the cecum six years after a choledochoduodenostomy. The patency of the common duct had been restored and no trace could be found of the anastomotic opening.

It is also of interest to emphasize the low mortality of this operation which has been strongly advocated by Sasse, Flörcken,[29] and Peterman as a means of providing internal drainage in place of a T tube or a buried tube after an ordinary choledochotomy. Sasse reports 46 cases without mortality, and of the 20 reported by Flörcken, only one died from peritonitis due to suture insufficiency. Sasse and Peterman call attention to the fact that this operation is contra-indicated by undue friability of the wall of the duct and by the presence of its cystic dilatation. External drainage is preferable in the presence of cholangeitis, of calculi or precipitated bile in either the common or hepatic duct. Flörcken adds the presence of ascarides, a narrow common duct, and beginning atrophy of the liver. Under all other conditions its low mortality and excellent results justify the general adoption of this operation which, hitherto, seems to have been employed solely by the above mentioned surgeons.

Formerly the ease with which duodenal contents could enter the duct after choledochoduodenostomy was thought to predispose to the danger of an infectious cholangeitis. Latterly, however, this fear has been dissipated by the fact that, after a choledochoduodenostomy, in numerous instances, barium, introduced into the stomach, has been found to penetrate to the hepatic tributaries, with no indication of even mild cholangeitis and, in the case reported by Hunt,[51] air in these same vessels was demonstrated roentgenologically three years after operation. It is also of interest to point out that duodenal contents have regurgitated through the opening in the common duct and have emerged through the abdominal incision after a simple choledochotomy. Codman was the first to report such an occurrence and latterly Walters[105] has added four other instances in which the ampulla was not subjected to any operative measure. In each of these five cases, however, the patency of the ampulla was demonstrated by the passage of a probe or of a uterine sound (Codman), and this may have caused temporary paralysis or relaxation of the sphincter of Oddi. Even, however, without instrumentation or any opera-

tion whatever, this sphincter is not always competent. Such an instance was demonstrated by my associate, Doctor Jennsen, who found the common duct distended with barium in an ordinary roentgenologic investigation of the duodenum.

Transplantation of Biliary Fistulae.—The writer has collected 41 cases of implantation of biliary fistulae into the stomach or intestine, from a search of the literature and from personal communications with surgeons here and abroad. Of these, 29 patients were operated upon in this country and six in Germany. In 18 the fistula was transplanted into the stomach, in one into the jejunum, in the remainder into the duodenum. The unfortunate experience in four cases observed respectively by von Stubenrauch,[99] Cahen,[13] Heidenhain, and Dobrotworski,[17] in which, after several weeks, a rapid contraction of the opening of the biliary fistula into the stomach was demonstrated either by relaparotomy or autopsy, has led these surgeons to conclude that implantation of the fistula into that organ should be avoided. In this country contraction of the biliary fistula proper has sometimes been observed, as in one of Walter's[106] cases, in which such a contraction developed four months after the implantation of a biliary fistula into the stomach, and a second time in the same patient, five months after the operation had been repeated. Also in Vincent's case, contraction of the biliary fistula followed its implantation into the duodenum. On relaparotomy, only a small probe could be passed through the fistulous tract which, after being dilated to the size of 14 F, was again implanted into the duodenum. The patient suffered from repeated attacks of cholangeitis and died three and one-half years after the first operation from cirrhosis of the liver with enlarged spleen. Vincent's comment on the value of this operation follows: "That a partial result that begets cirrhosis is not worth while. When possible I should endeavor to unite the mucous membrane of the duodenum to that of the divided duct in the hope that, if the patient survived this more formidable procedure, life would be worth while." In the cases reported by Lahey,[59] several failures were due to repeated attacks of cholangeitis with, in one instance, the filling of one-half of the abdominal cavity with an enlarged liver, the patient, subsequent to the operation, being invalided the greater part of the time. The occurrence of this unfortunate complication in cases reported by Lahey, Vincent, Walters,[107] Judd and others, proves that ascending infection may extend along the course of a biliary fistula and thence along the duct with which it communicates, to the parenchyma of the liver. Notwithstanding a brilliant result in which the patient was symptom free 15 years after the operation, Lahey comments rather pessimistically as follows: "It is my opinion that the operation is bound to be followed by a good many failures as it is really a makeshift procedure. More and more I become convinced as does everybody else that the procedure offering the best chance is, if possible, direct anastomosis of the duct." More encouraging results are reported by Russell,[88] whose patient was symptom free four and one-half years after operation, by St. John, whose patient was still symptom free when he was lost track of three years after

operation, by Lilienthal, whose patient died from cancer of the stomach four years after operation without any indication of biliary trouble, and by Roeder,[90] whose patient, at the end of seven years, suffered only from slight attacks of indigestion. Masson's patient died, symptom free, nine years after implantation, from cerebral hemorrhage, and Walter's three cases were in excellent health two, two and three-quarters, and three and one-half years respectively after operation. The most striking case of all is the patient of Hugh Williams who at present, 23 years after operation, enjoys perfect health. The fact that, in this case, the implantation was done when the patient was only four years old may account for the satisfactory epithelization of the biliary tract that must have taken place. In Lilienthal's case, in which, unfortunately, there was no autopsy, the implantation of the fistula with a collar of integument may have been a factor in the development of the malignant growth.

Abundant blood supply in the wall of the fistulous tract decreases the danger of its ultimate contraction. Its dissection, therefore, should not be carried beyond the free border of the liver and it should be only sufficiently long to insure anastomosis without undue tension. It should be coned out in such a way that its circumference increases as its deeper portion is exposed. A period of at least three months is essential to insure adequate vascularization of the wall of the fistula.

THE WILMS-SULLIVAN OPERATION.—In this country the credit of originating this operative procedure is quite properly given to Arthur G. Sullivan[96] who conducted his preliminary research in 1907. Abroad, where the operation has been more frequently employed, it is usually referred to as the "Wilms operation."[116] As a matter of fact Prof. Jenckel of Altoona should be given the chief credit, as he performed this operation in 1905 upon a patient aged 52, who still enjoys excellent health without the slightest evidence of recurrence.

The newly formed wall of the reconstructed duct must be composed, as far as is practicable, of immobile contiguous tissue. If the great omentum is employed for that purpose, only such portions as are not susceptible to the movement of the transverse colon, or likely to change position from pressure of adjacent viscera, should be utilized, for any movement whatever, if it does not actually endanger the integrity of its wall, may easily result in unfortunate angulation of the reconstructed channel.

Thirty-five cases are herewith reported. Of these, abstracts of the histories of five patients operated upon by Wilms, one by Jenckel, and one by Sullivan were presented by the writer in 1917. With few exceptions, the results are discouraging and more than one surgeon abroad has discarded the operation.

Unquestionably the most brilliant result was that obtained by Jenckell,[53] whose patient, reported in the Deutsche Zeitschrift, vol. 95, was alive and well, in full possession of her faculties, at the age of 82, 30 years after the operation. Another patient reported by the same surgeon suffered from

attacks of cholangeitis for six years after the operation, which invariably yielded to medical treatment. The attacks later on became more severe, although at one time, 11 years after the operation, the patient's condition was excellent, and she was free from jaundice. However, a relapse followed and the patient died a year later. On autopsy the liver was cirrhotic with an abscess in the right lobe. The reconstructed duct was patent, though somewhat narrowed. Professor Jenckel believes that death was due to the cirrhosis and its associated lesions. A third patient operated upon by Jenckel was symptom free at the end of four years, when he was lost sight of. Sullivan's patient died from cancer of the colon eight years after the operation of duct reconstruction. Hagler reports a case in which death occurred seven months after duct reconstruction by the passage of a tube from the hepatic stump into the duodenum, the cause of death being abscess of the liver.

It is therefore obvious that this method of duct reconstruction is also subject to both mild attacks of cholangeitis and to the more serious results of an ascending infection.

In Jenckel's brilliant case, a tube the size of an index finger was used. Although this may increase the possibility of a duodenal fistula (which actually occurred in this patient), it is quite probable that the use of a large tube insures a larger caliber of the reconstructed duct and thereby diminishes the chance of its subsequent contraction.

HEPATOJEJUNOSTOMY.—Nordmann[80] reports a case in which this operation was performed for a stricture following an immediate end-to-end anastomosis of a divided duct. The patient died three years later from septic cholangeitis. Nordmann also mentions four other cases of which three were well several years after the operation, the fourth dying of septic cholangeitis at the end of a year.

Enderlen[24] reports a case in which the patient was symptom free at the end of 11 years. In this instance the operation was supplemented by a Brauns anastomosis. Enderlen also reports a second case in which this operation, with the same modification, was done after the failure of a previous hepatoduodenostomy. An autopsy on the eighth day after operation disclosed an abscess of the liver, evidently of some months' duration, with a complete obliteration of the original anastomotic opening.

The seven cases in which this operation was performed are too few to permit drawing any definite conclusions as to its merit. Anastomosis of the duct to the jejunum is thought by many to increase the chance of subsequent serious cholangeitis. To minimize this risk Enderlen added a Brauns anastomosis, the stump of the hepatic duct being inserted into the extruded segment. The fact that cholangeitis does not always develop after this operation is demonstrated by the brilliant result in one of the patients reported by that surgeon, who was symptom free 11 years later. Hepatojejunostomy is indicated chiefly when the hepatic stump cannot be approximated to either the stomach or duodenum without undue tension. Under such conditions it is

considered preferable by some surgeons to reconstruct the duct by the Wilms' procedure, as well as to implant the biliary fistula.

ANASTOMOSIS OF THE ORIFICE OF A BILIARY FISTULA TO A SEGMENT OF JEJUNUM FROM WHICH THE INTESTINAL CURRENT HAS BEEN DIVERTED.—August Hildebrandt[45] suggested and carried out the following procedure. After division of the upper jejunum, intestinal continuity is reestablished by anastomosis of the oral segment to the jejunum at about 10 cc. below the point of division. The distal end of the divided jejunum is then brought out through the abdominal incision and after being passed through a tunnel behind the rectus muscle, is approximated to the mouth of the biliary fistula. A short tube is placed in the anastomotic opening and left in situ for four weeks. The patient upon whom this operation was performed died five years later in a condition of extreme icterus. Autopsy showed a large abscess beneath the liver into which both hepatic ducts opened. The abscess communicated with the intestine by a small channel, the opening in the gut being the size of the head of a pin.

Dobrotworsky[17] reports a case in which the loop of jejunum, after being placed within 2 cm. of the orifice of the biliary fistula, was temporarily closed. After ten days a small opening was made in it and a tube inserted joining the two orifices. Nine years afterward the patient was symptom free. He states that the "Patient attends to the fistula in the following way. Once a month the tube is removed and replaced by a duplicate. A small gauze bandage is sufficient to absorb the slight discharge. During the past year the mucous membrane of the jejunum slightly prolapsed with an increase in the amount of mucus. There has been no attack of cholangeitis during the entire period nor any tendency for the fistula to contract." He also reports a second case, symptom free after five years, in which peristaltic activity of the intestine tended to expel the connecting tube. This was relieved by the insertion of a longer drain.

Gonterman[38] (personal communication) writes that in one case in which the above operation was done the result was good. No further details were given. He also writes that a second patient died from strangulation of the loop brought out through the abdominal incision, due to angulation with consequent constriction of its blood supply, and warns against the occurrence of this accident.

Braeünig[119] (personal communication) describes a modification of this procedure as follows: After reestablishing intestinal continuity in the above manner, the biliary fistula is coned out and anastomosed to the open orifice of the divided jejunum. Recurrence of the symptoms of stricture in a patient subjected to this procedure occurred a half-year later. On relaparotomy, complete closure of the biliary fistula was found together with a large accumulation of bile in the retroperitoneal epigastrium. Duct reconstruction over a T tube between the upper choledochus and the duodenum was accomplished. Twenty-one months later patient was symptom free.

ANASTOMOSIS OF THE HEPATIC OR COMMON DUCT TO THE STOMACH.—

Dr. Paul Tschassownikoff[97] of Odessa reports a case of anastomosis of the hepatic stump over a small tube with the pylorus, preceded by denudation of a pyramidal shaped area in the adjacent liver to promote, by adhesion, a barrier against leakage. This patient was symptom free 14 years after operation (personal communication).

Schweizer:[94] Recurrence followed the above operation within a year, due, he thinks, to the too early passage of the tube (five weeks). The operation was repeated over a T tube which remained in situ for a year. During this time there was occasional bleeding around the tube, which was replaced by a catheter. Death occurred from hemorrhage three years after the second operation.

Walzel:[111] Hepatogastrostomy with tube brought through stomach wall (Witzel). Repeated five times in two and one-half years for recurrence: Death from nephritis and inanition.

Lewis[120] reports a case with an immediate satisfactory result of anastomosis between the orifice of a biliary fistula in the stomach and the hepatic stump. Sufficient time has not elapsed to determine the end-result.

Choledochotomy for Stricture.—Schweizer and the writer report respectively cases of this operation for the relief of stricture. In Schweizer's case the patient was symptom free 14 months after operation and in the writer's case the patient was lost track of about 18 months after operation. There had been in the interval one mild attack of cholangeitis. Mere division of a benign stricture temporarily relieved obstruction. Were subsequent dilatation possible the result would be most satisfactory. Recurrence of the stricture, however, can be prevented only by the insertion and retention of a rubber tube. A stricture at the ampulla was divided by Fründ from within the duodenum and a rubber tube inserted which remained in situ for two years, during which time the patient suffered from attacks of cholangeitis. On relaparotomy the duct was found widely distended above an incrusted tube, which was removed and a choledochoduodenostomy performed. Nine years later the patient was symptom free.

Postoperative complications include hemorrhage, shock, peritonitis, due either to leakage or to infected bile, acute pancreatitis, subdiaphragmatic abscess, septic cholangeitis with abscess of the liver, septic thrombosis of portal radicals with or without mesenteric thrombosis, and finally terminal cirrhosis with occasionally splenic involvement. The presence of noninfected bile in the peritoneal cavity is dangerous only when it distends the lesser peritoneal cavity or when it accumulates between the dome of the liver and the diaphraghm (Leriche and Walters) with consequent pressure downward on the liver and interference with the flow of blood through the hepatic vein. Accumulation of noninfected bile in the greater peritoneal cavity is well tolerated. J. F. Erdmann removed several gallons 14 days after cholecystectomy; a fortnight later a successful choledochoduodenostomy was performed. The writer also recalls the accumulation of a large amount of biliary ascitic fluid after a subcutaneous rupture of the liver, which was removed by aspiration two weeks

after the receipt of the trauma. In neither case was the condition of the patient serious and in both recovery promptly ensued.

Accurate estimate of the relative value of these different operative procedures is impossible. In general the selection of the more simple operation is indicated. End-to-end anastomosis, when practicable, after excision of the stricture, affords an excellent chance of success. In strictures of the common duct, choledochoduodenostomy, or hepatoduodenostomy, especially where the mucous membrane of the duct can be approximated to that of the stomach or intestine without tension, is evidently the operation of choice. Duct reconstruction by the Wilms' method has not usually given encouraging results. Implantation of biliary fistulae into the stomach or duodenum appears preferable. Either one or the other of these two procedures or an anastomosis of the duct with the jejunum must be attempted when the greater part of the hepatic and common ducts is obliterated. Treatment of strictures of the hepatic duct within the liver still present a most difficult problem. An attempt should be made to establish a fistula with the dilated portion of the duct or with a segment of liver parenchyma, previously penetrated with the cautery, which subsequently may be transplanted into the stomach or duodenum. The few attempts of anastomosis of the duct or fistula with a loop of jejunum, from which the intestinal current has been diverted, do not justify an estimate of the value of this particular method of treatment.

In conclusion it is a privilege and pleasure to acknowledge the kindness of colleagues abroad and in this country for their painstaking cooperation. Without these courtesies the preparation of this paper would have been impossible.

Abbreviated Case Reports

WILMS' OPERATION

Wilms-Brandt:[116] Entire common duct obliterated. Well 15 months later.

Wilms-Brandt: Tube from hepatic stump into duodenum. Well 15 months later.

Wilms-Brandt: Tube from hepatic stump into duodenum. Well one and one-half months later.

Wilms-Brandt: Tube from hepatic stump into stomach. Well six months later.

Wilms-Brandt: Tube from choledochus into duodenum. Well one and one-half months later.

Hagler:[44] Seven months after reconstruction with tube, death occurred from abscess of the liver.

Naegele:[78] Tube from duct into duodenum followed in several weeks by stenosis of duodenal orifice, relieved by plastic. No end-result mentioned.

Magnus:[68] Reports two operative deaths.

Simon:[93] Reports a case complicated after operation by abscess around the choledochus. Prefers direct anastomosis of duct to stomach or duodenum.

Enderlen: Reports an operative death.

Feist:[33] Gap 4 cm. long bridged by tube from choledochus into duodenum. Symptom free one year after operation; then (personal communication) recurrence with swollen liver and probable death.

Sullivan: Reconstruction with tube for stricture 1½ cm. long. Died eight years later from cancer of the cecum without sign of recurrence.

Jenckel: Gap 7 cm. long bridged by tube, the size of index finger, from hepatic duct into duodenum. Mild cholangeitis for several months, then symptom free. Patient, now aged 82, is well 30 years after operation.

Jenckel: Gap 7 cm. long bridged with tube. For six years postoperative had mild attacks of cholangeitis, which yielded to medical treatment. Eleven years after operation symptom free. Then jaundice with death a year later. On autopsy the duct was patent though somewhat narrowed. There was an abscess in the liver. Cirrhosis, to which Jenckel ascribed the death of the patient. No microscopic examination of the duct was made.

Jenckel: Gap from duct divided during cholecystectomy was bridged for a distance of between 3 and 4 cm. Stricture formed after six months. Relaparotomy disclosed the tube between a cystic dilatation above and the choledochus below. Death occurred from biliary peritonitis.

Jenckel: Gap of 3 cm. during cholecystectomy. Bridged by tube. Four years later, when patient was lost sight of, there was no sign of recurrence.

Erdmann, J. F.:[26] Gap from division of the duct during cholecystectomy. Bridged by tube which passed in five weeks. One year later recurrence of stricture was relieved by hepatoduodenostomy. Patient well five years later.

Jenckel: Refers to six additional cases in which this operation was performed without a single failure.

Whipple: Wilms' operation bridging a gap between the hepatic duct and duodenum. Patient relieved for four months. Then recurrence. Subsequent operation failed to reestablish the duct continuity into the duodenum. Patient died two years after the original operation.

Franz, C.:[31] Reports two cases followed by stricture.

Brin, H.:[7] Tube bridging hepatic stump and duodenum. Condition good one year later.

Savariaud:[92] Tube bridging hepatic stump and stomach. One year later condition fair. Some attacks of cholangeitis.

Gernez:[37] Tube bridging hepatic stump and stomach. Recovery. No end-result given.

Tuffier: After tube operation symptom free for two years. Then progressive cholangeitis with death notwithstanding a biliary fistula.

Lobmeyer:[62] Tube bridging hepatic stump and duodenum. No end-result given.

Hübsch:[50] Tube bridging hepatic stump and duodenum. No end-result given.

Walzel: [112] Tube bridging gap of 4 cm. Well two and one-half years later.

Walzel: Tube bridging hepatic stump and duodenum. Eleven months later the tube, blocked and foul, was removed.

Walzel: Four months after treatment of stricture of choledochus with T tube, recurrence. Gap of 2 cm. was then bridged by long tube extending into duodenum. Recurrence seven months after was relieved by hepatoduodenostomy. Symptom free eight months later.

REPAIR OF DUCT OVER BURIED TUBE

Steindl: End-to-end suture over a rubber tube of common duct defect, 3 cm. in length, tube projecting not over one inch into the lumen of the duodenum. Excellent result. Time not stated.

Steindl: Sixteen months after instituting hepatic drainage in extensive stricture of the choledochus, tube passed from hepatic stump into duodenum and covered with contiguous tissue (Wilms-Sullivan operation). Recovery.

Steindl: Defects in the wall of both common duct and duodenum each 1½ cm. in diameter were repaired over a rubber tube. The consequent constriction in the duodenum was relieved by a posterior gastro-enterostomy. Excellent result. Time not stated.

Whipple: After repair of common duct over a buried tube, patient lived for two years and then died from biliary cirrhosis with enlarged spleen.

BILIARY FISTULA IMPLANTATION

Russell:[88] Biliary fistula of seven months' duration into the stomach. Well four and one-half years later.

St. John: Fistula with skin encircling orifice into stomach. Patient symptom free at end of three years, when lost track of.

Lilienthal: Biliary fistula with skin encircling orifice into stomach. Patient died four years later from cancer of stomach without sign of recurrence.

Williams: Fistula in patient aged four into duodenum without tube. Patient symptom free 23 years after operation.

Roeder: Fistula into duodenum. With the exception of occasional fat indigestion patient symptom free seven years later.

Roeder: Biliary fistula into duodenum using a long catheter. This subsequently became disengaged and caused death by penetrating the pelvic sigmoid, three months after operation.

Masson: Fistula into duodenum. Death on fourth postoperative day from hemorrhage.

Judd: Fistula into stomach. Death on third day from shock.

Judd: Fistula into stomach; one and one-half years later, recurrence, relieved by hepatoduodenostomy with dilatation of the stricture.

Pemberton: Fistula into duodenum. Four months later contraction of the fistula. Duct reconstruction over a T tube.

Walters:[108] Fistula into duodenum. Well two years later.

Walters:[109] Fistula into stomach. Death fourth day after operation.

Walters:[110] Fistula into duodenum. Well two and three-quarter years later.

Judd: Fistula into duodenum (?). For following two months intermittent jaundice. Death three years after operation.

Walters:[104] Fistula into duodenum. Well three and one-half years later.

Walters: Fistula into stomach. Death fourth day from hemorrhage and hepatic insufficiency.

Masson: Fistula into duodenum. Patient died nine years later from cerebral hemorrhage without sign of recurrence.

Walters:[106] Fistula into stomach. Four months later, contraction. Operation repeated followed by a second contraction nine months later. Patient died five months after the second operation.

Walters:[107] Fistula into stomach. Too recent for end-result.

Vincent: Fistula into duodenum. Intermittent attacks of cholangeitis 14 months after operation; fistula had contracted to fibrous cord. It was dilated to 14 F. and again anastomosed to duodenum. Attacks of cholangeitis continued and patient died three and one-half years after first operation from enlarged liver and spleen.

Lahey:[59] Fistula into stomach. For several months cholangeitis. Then symptom free and well after 15 years.

Lahey: Fistula into duodenum. Periodic attacks of cholangeitis. Liver greatly enlarged filling more than one-half of the abdomen.

Lahey: Fistula into stomach. Then cholangeitis and a subdiaphragmatic abscess which was drained. At end of three years, when patient was lost sight of, condition was unsatisfactory.

Lahey: Fistula into stomach. Bedridden with cholangeitis. No recent report.

Lahey: Fistula into stomach. No cholangeitis. No recent report.

Lahey: Fistula into duodenum. Death three months from cancer of the pancreas.

Lahey: Fistula into duodenum. Death three months after operation.

Lahey: Fistula into duodenum. Death 13 days later from persistent hemorrhage.

Lahey: Death on fourth day after fistula into duodenum, subsequent to a previous gastrectomy.

Heidenhain: Fistula into stomach. Contraction in three weeks, followed by tube reconstruction into duodenum. Cholangeitis and death (probably) three years later.

Tietze:[98] Fistula into jejunum. Death from abscess of the liver.

Magnus: Fistula into duodenum. Well two months later.

Von Stubenrauch, Cahen, and Dobrotworsky have all experienced contraction of fistulae into stomach.

Whipple: Implantation into duodenum. Operative death.

Whipple: Implantation into duodenum. Operative death.

Whipple: Implantation into duodenum. Operative death.

Whipple: Patient well 55 months after operation.

Whipple: Implantation into stomach followed by stenosis. One year later choledochoduodenostomy. End-result not stated.

Pickhardt: Implantation into duodenum. Patient gradually failed and died after several months.

HEPATODUODENOSTOMY

Von Redwitz: Symptom free two years after operation in which the tube passed through a Witzel opening in the duodenum, was brought out of the abdominal incision. (Posterior gastro-enterostomy.)

Leriche: Recurrence two months after duct reconstruction with buried tube (due possibly to the too early passage of the tube). Although nine months later there was an accumulation of bile over the hepatic dome, patient was symptom free for the following 21 months after the hepatoduodenostomy except for two mild attacks of cholangeitis. The attacks then became more intense and patient died five years after the anastomosis.

Moritz Cohn: Hepatoduodenostomy with tube emerging through Witzel opening in the duodenum, and which was removed on the twelfth day. Well 12 years after operation.

Melchior: Death between one and two years after operation from liver abscess (see end-to-end anastomosis).

Alessandri:[1] Operation for extensive stricture. Tube was removed through an incision in the duodenum at the end of a year. Patient symptom free eight and one-half years after operation.

Wallstein:[118] Operation over tube extending upward into both left and right hepatic ducts (tending to keep tube in place). Well three years later.

Enderlen: Hepatoduodenostomy owing to a complete obliteration of the orifice of a previous jejunoduodenostomy. Died eight days later.

Bernhard:[9] Reports 13 cases with five operative deaths. One patient died four years after operation from septic cholangeitis, three have occasional attacks of mild cholangeitis and three are symptom free, 10, 11, and 24 years after operation.

Erdmann, J. F.:[25] Hepatoduodenostomy performed shortly after the evacuation of a large amount of bile from the abdomen. Symptom free 13 years after.

Erdmann, J. F.:[26] After failure of Wilms' operation, hepatoduodenostomy over tube, remaining in situ four months. Symptom free 17 years later.

Erdmann: Recurrence of symptoms of stricture due to the clogging of the tube over which the anastomosis was done five and one-half months after operation. Relieved by inserting a smaller tube. Final result not known.

Erdmann: Death from pneumonia one year after operation, in a condition of extreme jaundice.

Erdmann: Save for occasional mild attacks of cholangeitis and pain, patient is symptom free 18 years after operation.

Erdmann: Recurrence, patient being subsequently operated upon by another surgeon.

Verdi:[101] Ten months after an end-to-end anastomosis, hepatoduodenostomy for recurrence. Nine months later the calcified tube was removed through an incision in the duodenum. Since then symptom free, ten years after operation.

Verdi: Recurrence eight months after hepatoduodenostomy, patient dying subsequently from pneumonia, unrelieved.

Hunt: Hepatoduodenostomy over a tube (passed five months later). Several mild attacks of cholangeitis. Symptom free at end of five years. Recent roentgenograms show passage of air into the tributaries of the hepatic vein.

Fowler:[42] Recurrence after a few months due probably to the too early passage of the tube. Operation repeated by another surgeon with death three years after the first operation in jaundice.

Walters:[108] Refers to 15 cases of either hepato- or choledochoduodenostomy without details. Of these, one was symptom free at the end of five years, two at the end of two years, and two at the end of one year.

Mayo, W. J.: One of the earliest operations performed (1904). Patient symptom free 15 years later.

Judd: Cholangeitis four years after operation. Tube was removed six years after operation. Patient symptom free six years after operation.

Judd: Operation followed by recurrence of stricture in one year. Operation repeated over a smaller tube. Well one and one-half years after.

Judd: Operation followed in ten months by cholangeitis. Death four years after operation from biliary cirrhosis.

Guerry: To avoid tension a gap of half an inch, at the anastomosis, was bridged by small rubber tube which was passed on the fourteenth day. Symptom free 19 years after operation.

Guerry: Patient symptom free five years after operation. Lost sight of.

Guerry: Well at present (70 years old), 12 years after operation.

Guerry: Symptom free eight years after operation.

Guerry: Symptom free six years after operation (now over 70).

Guerry: Symptom free three years after operation; lost sight of.

Guerry: Symptom free five years after operation.

Walzel: Reports seven cases, of which the first died after several operations from extensive yellow atrophy of the liver. This led him to devise a special technic (see original article) in the remaining six. Of these, two succumbed to operation, the remaining four being symptom free for various periods up to three years (1929). (Inquiry as to the present condition of these patients has failed to elicit any response.)

Whipple: Patient died 18 hours after operation.

Whipple: Patient symptom free 20 months after operation.

Whipple: Patient symptom free 56 months after operation.

Dreesman:[20] After failure of end-to-end anastomosis a hepatoduodenostomy, supplemented by posterior gastro-enterostomy with division of the pylorus, was performed. Recovery. No end-result mentioned.

END-TO-END ANASTOMOSIS

Von Redwitz: Suture of duct three days after it had been divided during cholecystectomy. Recurrence in less than a year and again after a second operation in ten months. Then hepatogastrostomy, the hepatic stump being sutured to the edge of a gastric defect, due to the pressure of the rubber tube, supplemented by a posterior gastroenterostomy. Symptom free one year later.

Melchior: Stricture following an immediate suture of the divided duct during cholecystectomy, then hepatoduodenostomy. Death from abscess of the liver between one and two years later.

Braeünig[119] Suture of duct divided during cholecystectomy with tube extending through the duodenal wall (Witzel). Complete biliary fistula. Three months later suture of fistula to the divided end of excluded jejunal loop.

Tietze:[98] Subsequent stenosis and death, in another hospital, after end-to-end suture over a T tube for stricture.

Papin:[84] Suture of partially divided duct during cholecystectomy without a tube. Symptom free ten years later.

Fiolla: Symptom free for two years after suture of divided duct ends. Death from violent septic cholangeitis.

Downes:[19] End-to-end suture of duct divided during cholecystectomy. Death from cancer of the prostate 16 years later.

Downes: Division of stricture subsequent to cholecystectomy one-eighth of an inch long with T tube for a month. Operation repeated for recurrence two months later. Subsequent cholangeitis for two or three years, gradually decreasing. Since then mild occasional bilious attacks from indiscretions in diet. Otherwise symptom free 13 years after operation.

Downes: Well for 28 months after suture of duct divided during cholecystectomy. Then cholangeitis followed by drainage with a T tube for seven months. Death one year later from mesenteric thrombosis and cirrhosis.

Douglas:[18] Suture over a T tube for stricture between 1 and 2 cm. in length. During the following year attacks of cholangeitis. Then symptom free. Well 11 years later.

Douglas: End-to-end suture for stricture following division of the hepatic duct near cystic duct during cholecystectomy. Cholangeitis attacks during the second year after. At end of 14 years symptom free.

Matthews:[65] End-to-end suture for stricture, months after a cholecystectomy, over tube emerging through an opening lower down in the duct. Well 14 years.

Matthews: End-to-end suture of the hepatic duct divided during cholecystectomy, a catheter being inserted down through the stump of the cystic duct. Early removal of the tube. Some cholangeitis for a year. Well four years.

Nordmann:[80] Immediate suture of divided duct. Recurrence of stricture, then hepatostomy.

McArthur:[75] Suture over tube with "reversed cuff" extremity after excision of stricture following cholecystectomy. Tube passed on sixty-third day. Death from cancer of the stomach two years later.

McArthur: Suture after excision of stricture over "cuffed" tube which never passed. Death from cholangeitis 18 months later.

McArthur:[76] Recurrence four years after suture for stricture. Then choledochoduodenostomy over tube which passed on twenty-seventh day. Occasional attacks of cholangeitis. Patient well six years later.

McArthur: Suture of duct over "cuffed" tube which passed nine weeks later. Recurrence. Operation by Doctor Finney, patient dying one year later in jaundice.

McArthur: Suture of duct over "cuffed" tube. Well five years later.

Seward Erdman:[27] Immediate suture of duct divided during cholecystectomy over tube which became clogged three months later. Smaller tube inserted. Nine months after operation cholangeitis, repeated since with decreasing frequency. In good condition three years after operation.

Bartlett, W:[43] Suture over a T tube for stricture in common duct. Tube in place one year. Symptom free at end of six years.

Schweizer:[94] Suture of duct ends over a T tube which remained in place for 18 months. Symptom free at end of 14 years.

Riggs:[89] End-to-end suture for gap of 3 cm. after removal of cicatricial tissue. Death from myocarditis 14 years later, without recurrence.

Stetten:[95] End-to-end suture of hepatic duct. Thirteen years later mild attack of cholangeitis due probably to a cardiac condition, from which patient died without evidence of recurrence 19 years after operation.

Homans:[47] End-to-end suture of duct completely torn across during cholecystectomy. Symptom free at end of ten years.

Homans: Division of common duct with nicking of the hepatic duct during chole-

cystectomy. Immediate suture. Gallbladder left in situ. Symptom free ten years after operation.

Walters:[108] Four cases of end-to-end suture after excision of stricture. Two well one year after operation, the others for shorter periods.

Horgan:[49] End-to-end with L shaped tube, the extremity of which was brought out through a separate incision in the wall of the duct. Symptom free seven years.

Horgan: Sutured after excision of a stricture in the hepatic over L shaped tube brought out through a separate incision in the wall of the duct. Symptom free at end of eight years.

Horgan: Suture of choledochus torn across in the delivery of a stone during choledochotomy, over L shaped tube brought out through separate incision in the wall of the duct. Symptom free at end of ten years.

Judd:[54] End-to-end over T tube after excision of stricture. Well five years later.

Judd: End-to-end followed by abscess of the liver. Drainage. Subsequent choledochoduodenostomy.

Judd: Fifth recurrence within a year after end-to-end suture. Reconstruction of duct. Well two years after.

Judd: End-to-end suture over T tube. Well seven and one-half years later.

Judd: Gap resulting after excision of stricture in hepatic duct filled by interposition of the cystic stump. Well seven years later.

Balfour:[4] End-to-end anastomosis of hepatic duct divided during cholecystectomy. Patient well five years later.

Eliot:[22] End-to-end suture months after cholecystectomy for stricture in common duct. Death 18 years later from intercurrent disease.

Eliot:[23] End-to-end suture of hepatic duct months after cholecystectomy. Several attacks of cholangeitis following operation. Symptom free ten years after.

Walzel:[117] Divided ends of choledochus sutured over a long tube into duodenum. Well two and one-half years later.

CHOLEDOCHODUODENOSTOMY

Downes:[19] Four weeks after hepatic drainage, choledochoduodenostomy over a 14 F. catheter. Nine months later recurrence. Dilatation of stricture through duodenal approach up to 18 F. with insertion of tube. During following year attacks of cholangeitis of increasing frequency and severity with death six years after the first operation and five years after the dilatation.

Von Redwitz:[100] Choledochoduodenostomy three weeks after cholecystectomy for stricture of common duct. Three years later hepatoduodenostomy over tube introduced after the Witzel method into the duodenum, with posterior gastrostomy. The previous anastomosis was obliterated.

Sasse:[91] Choledochoduodenostomy for stricture, the duct containing purulent material. Successive attacks of first mild and later severe cholangeitis until the death of the patient six years later. Autopsy showed calculi in the duct, a lumbricoid, abscess of the liver and basilar pneumococcus meningitis.

Behrend:[5] On the twenty-first day after a choledochoduodenostomy for stricture, a T tube was inserted into the duct because of jaundice, draining bile and duodenal contents. Symptom free 19 years afterward. Behrend refers to "several similar cases since, all symptom free."

Williams: Choledochoduodenostomy for acholic stools. Symptom free three years later.

Brooks:[11] Choledochoduodenostomy (blind end of duct). Well one and one-half years afterward.

Lewis:[120] Two cases of choledochoduodenostomy (blind end of duct). Too recent for end-result. Postoperative cholangeitis in one case.

Peterman:[85] Choledochoduodenostomy for stricture at the ampulla (possibly inflammatory). Well 12 years later.

Judd:[54] Choledochoduodenostomy following failure of end-to-end anastomosis over a tube, which was vomited up a year later. In following year abscess of liver, successfully drained. Then well three years later.

Walters:[110] Choledochoduodenostomy for stricture 1½ cm. long. Symptom free after two years.

Fensterer: Choledochoduodenostomy for stricture over a rubber tube. Patient died six years later from cancer of the cecum. Autopsy showed a patent choledochus with complete obliteration of the anastomosis.

Guerry:[41] Choledochoduodenostomy. Death from ascending infection four years after operation.

Guerry: Two cases dying from shock and pneumonia, shortly after operation.

Guerry: Choledochoduodenostomy. Symptom free six years later.

Guerry: Choledochoduodenostomy for duct injured during choledochotomy. Symptom free one year later.

Cave:[14] Choledochoduodenostomy with Murphy button for stricture in common duct. Patient symptom free nine years later.

Whipple: Choledochoduodenostomy. Death from peritonitis four days later, due to tearing of suture line.

Whipple: Choledochoduodenostomy. Death on sixth day after operation from periduodenal hemorrhage causing obstruction.

Whipple: Choledochoduodenostomy. Symptom free two years later.

Whipple: Choledochoduodenostomy. Well for 30 months, then one attack of jaundice with death three months later. Death reported as carcinoma, not proved by autopsy.

Fründ, H.:[34] Rubber tube inserted through papilla (transduodenal) up into and through stricture of common duct. During following two years, cholangeitis. Then duct found widely dilated above stiffened and incrusted tube. Choledochoduodenostomy tube being removed. Symptom free nine years later (aged 61).

Kaspar: Reports six cases of cicatricial stricture of which four were associated with calculus in the common duct. Those without calculus had followed a previous cholecystectomy. Of 38 cases of choledochoduodenostomy for various conditions, only one patient died (cancer of pancreas; pneumonia). Like Sasse and Flörcken, Kaspar is an enthusiastic advocate of this operation and thinks it is vastly superior to Kehr's external drainage. Of 99 cases (Jurasz, Toole, Sasse, Flörcken, Peterman, Moll, Lowenstein, von Haberer, and Kaspar) operated upon for various conditions, only six deaths occurred. In only three instances was opportunity afforded to subsequently examine the site of anastomosis. In Toole's case (nine months after operation), and in Jurasz' case (13 months after operation), the anastomotic orifice was patent. In Sasse's case (six years after operation), the orifice was completely closed. Kaspar cites the following advantages over Kehr's external drainage: Prevention of loss of bile, no pressure ulceration, no angulation of tube, no persistence of the fistulae.

CHOLECYSTOGASTROSTOMY

R. T. Miller, Jr.:[73] For stricture common duct (no previous operation). Well one year later.

CHOLECYSTODUODENOSTOMY

Anschütz:[2] For stricture choledochus. Well 11 years later.

Whipple: For stricture in the common duct, following trauma, gallbladder and cystic duct being normal. Patient symptom free 70 months later.

Walters:[109] Cholecystoduodenostomy. Good result. Symptom free three years later. Barium seen to enter gallbladder.

Judd:[54] Cholecystoduodenostomy after two previous operations for biliary fistula, fol-

lowed in three months by cholangeitis. Subsequent reoperation disclosed stricture of the common duct for which hepatoduodenostomy was performed over a rubber tube, the stricture being excised. Cholangeitis developed ten months later and the patient ultimately died of biliary cirrhosis four years after the last operation.

Wolff:[117] Reports a case of anastomosis between gallbladder and choledochus. Well four years later.

CHOLEDOCHOCHOLECYSTODUODENOSTOMY

Whipple: Between a stricture following cholecystectomy (?) and the duodenum, a cavity resembling the gallbladder, and filled with bile, was interposed. This communicated with the dilated duct above the stricture. It was probably a newly formed adventitious space in the situation of the previously removed gallbladder. The utilization of a portion of its wall in forming the anastomosis accounts for the name of this unusual operation.

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Discussion.—Dr. Frank H. Lahey (Boston, Mass.).—Everyone who has been interested in strictures of the common and hepatic ducts owes Doctor Eliot a debt of gratitude. His original contributions in which the work done on anastomoses for strictures of the common duct, going over the literature up to approximately 1922, 1923, and 1924, as I recall it, covered all of the foreign and English speaking literature in the world, and is an extremely valuable article for anyone to read who is interested in this subject.

There is little to be added to what he has said except my own personal and practical experience with this subject. We have now operated upon 20 strictures of the common duct, and it is but fair to say that they have all been sent to us and are not cases we have produced ourselves.

One deals with three types of stricture. One is the hopeless type, in which an infection produces a fibrosis of the entire main biliary duct. This type we have found entirely hopeless, since this fibrosis extends up into the intrahepatic portion of the duct. A very hopeful type is one which occurred in a case we have just sent home, *i.e.*, with the stricture at the lower end of the common duct. I would call attention to the fact that these cases usually occur after cholecystectomies, that there is a dilation of the stump of cystic duct which can be implanted into the duodenum very satisfactorily. I believe in this type of stricture that this is more satisfactory than the lateral anastomosis between the dilated duct and the duodenum. The use of a small tube in the dilated cystic duct which can be implanted and sutured into the duodenum makes it a very satisfactory procedure.

Strictures of the hepatic duct in the majority of instances follow technical difficulties with the cystic artery. They are the result of excitement, I think.

The cystic artery gets loose, bleeds into the deep operative field. The inexperienced operator fails to realize that by pinching the hepatic artery with his finger he can stop the bleeding artery, wipe the field dry, find the bleeding vessel and control it accurately. Instead of this, he clamps wildly and picks up the hepatic duct in the clamp, thus causing the stricture. These are difficult types of stricture and I believe it requires the nicest judgment to determine whether one would perform a direct anastomosis of the duct or a reconstruction of it. We have in some of the cases, effected a Mikulicz type of reconstruction on the strictured ducts, only to have most of them become strictured again, and in some of these cases, I am sure it would have been better had we cut the strictured duct off and performed a direct anastomosis between the end of the duct and the duodenum. The most satisfactory operation in strictures of the hepatic duct will be the direct anastomosis between the end of the duct and the duodenum as suggested by Dr. William J. Mayo. I think the implantation of the external biliary fistulae will result in failure in most of the cases. We have performed 14, and only two have been permanently relieved, due to the fact, I think, that the fistulae in most cases contract and reobstruct.

There are some things which I think of practical value in connection with the implantation of the fistulae if one has no other recourse. If one has such a high stricture that it is close to the division of the hepatic duct, then there is nothing left to do but to implant the fistula. From our experience, I believe it is best to implant it into jejunum. If it is implanted in the stomach, it can easily be pulled out following vomiting and the vigorous gastric contractions associated with it. Furthermore, if implanted in the stomach, it will be pulled over toward the midline, thus making considerable traction on the fistula, and predisposing to necrosis of it and allowing it to be pulled away from its bed in the liver.

On the other hand, a jejunal loop can be brought up to the portion of the fistula which is attached to the liver and an implantation effected. The jejunum can then be sutured to the capsule of the liver so that there is no strain, upon the fistulous tract itself. These measures will, in our experience, produce better results in the implantation of fistulae but as already stated, the operation is in itself, of necessity, a poor one and a makeshift.

I think as to the treatment of strictures of the bile duct, we have accomplished more today, in voting in favor of certification of surgeons, than has been accomplished up to date by any of the proposed technical measures.

Dr. Allen O. Whipple (New York, N. Y.).—Almost all of the studies that have been made in the problem of bile duct reconstruction, as brought out so ably in Doctor Eliot's paper, carry through them the constantly recurring and sinister motif of cholangeitis. The prevention of this duct and liver infection seems to me to be one of the most difficult problems that we are faced with in the care of patients with duct reconstruction, where the reconstruction has to be done in order to save the patient from the misery and ultimate outcome resulting from complete stenosis.

As has been brought out by Doctor Eliot and Doctor Ivy in the discussion, the maintenance of the sphincter mechanism is the underlying reason for the success of the end-to-end anastomosis of the two cut ends of the duct. It is the lack of such a mechanism that is one of the real difficulties in the operation reported by us a year ago for radical removal of a part of the duodenum and pancreas for carcinoma of the papilla of Vater. In the paper read by Doctor Parsons last June he brought out the fact that in one of our cases the cholangeitis was the cause of death seven months after the operation, as a result

of multiple abscesses of the liver following cholangeitis. Since that time we have carried out the procedure in two other cases successfully; in one patient there have been transitory attacks of cholangeitis, but the patient is still living, 18 months after the operation.

If some means could be devised to avoid the occurrence of cholangeitis, I would be very much more enthusiastic about our procedure as a method of dealing with carcinoma of the papilla, but I wish to sound a note of warning in regard to the danger of cholangeitis where the gallbladder is anastomosed directly to the stomach, into which jejunal contents are emptying.

Dr. Waltman Walters (Rochester, Minn.).—In 1931,[1] I presented a group of 30 cases in whom resection of the common and hepatic bile ducts and ampulla of Vater had been performed by me at the Mayo Clinic. Since that time I have operated upon an additional 17 patients, making a total of 47. I had hoped to summarize all of these cases to date, but am sorry that circumstances have prevented the completion of this review, so it will have to be presented in detail at a later time. For the purposes of this discussion, I shall confine myself to a few general comments on the results we have obtained.

When there was sufficient duct above the stricture to enable one to anastomose it to an opening made in the duodenum, choledochoduodenostomy or hepaticoduodenostomy, this has been the preferable procedure, and it has been performed in 24 of the cases, in 11 during the period from 1932 to 1936.

Time permits only the briefest reference to the results in this series of 47 cases. Generally speaking, I have had no reason to change the opinions which I expressed in the conclusions published in 1933,[1] namely, that very good results have been obtained in many cases in which accurate anastomosis has been made between a remaining portion of the duct and an opening made in the duodenum. Failure to obtain lasting good results could almost be predicted at the time of anastomosis if the stump of the duct was too short to permit accurate mucous membrane-to-mucous membrane anastomosis to the opening in the duodenum, and in those cases in which it was evident that marked cirrhosis or intrahepatic infection was present, which was characterized by purulent bile or granular stones within the hepatic ducts. For example, in Cases 1 and 2 of the 1933[1] report, the patients are living and well without having had a return of evidences of biliary obstruction. In the first case ten years have elapsed since operation, and in the second, eight years. In both instances there was sufficient duct above the stricture so that an accurate anastomosis could be made between it and an opening made in the duodenum.

I formerly held the opinion that excision of a localized stricture of the common and hepatic ducts, in which anastomosis was made between the ends of the duct after the excision, was likely to be followed by recurrence of the stricture at the point of the anastomosis. However, contrary to this was the case (Case 18 of the 1933[1] series) in which I excised a neurofibroma that was obstructing the common duct at the bifurcation of the hepatic ducts. The two ends of the hepatic ducts were sutured to the end of the common duct. The present condition of this patient was recently reported by Comfort and myself.[2] Whereas she had occasional biliary colics the first year or two subsequent to operation, in the last four years she has been free of biliary colic and jaundice. In another instance (Case 16) local excision of a stricture of the common duct located directly beneath the liver has been followed by results which persistently remained good after a five year period.

Transplantation of external biliary fistulae, established because of complete

absence of an extrahepatic duct, continues to be a useful procedure. One of the patients who underwent this procedure in December, 1929 (Case 23), continues to be well and is without evidences of biliary colic or obstruction. That case was one of four in which I transplanted external biliary fistulae. I used the procedure in two other cases, in one recently in which, at the time the external biliary fistula was established, the patient was very deeply jaundiced and had a serum bilirubin of 13.5 mg. per 100 cc. In this case the patient had such a hemorrhagic tendency that in addition to subcutaneous hemorrhages, she also had subconjunctival, intestinal and uterine bleeding; the coagulation time was elevated to 16 minutes. After a prolonged period of preliminary preparation, consisting of the intravenous glucose, blood transfusions, and the administration of calcium, an external biliary fistula was established. The serum bilirubin rapidly decreased to 5.8 mg. per 100 cc. On March 27, 1936, I coned out and transplanted the fistula into the stomach. The incision healed promptly and she was allowed to return home April 24, 1936, with a serum bilirubin of 2.8 mg. per 100 cc.

SUMMARY.—Sufficient data are available in a group of 47 cases of stricture of the common duct, that were operated on by me at the Mayo Clinic, to show that plastic operations for stricture of the common and hepatic ducts are justified, not only from the standpoint of relief of obstructive jaundice, but prolongation of life.

When sufficient duct remains above the stricture so that an accurate mucous membrane anastomosis can be made to the duodenum, good results may be expected, providing there is not too much infection within the liver or in the wall of the stump of the duct used to make the anastomosis. Many such instances of this type have been reported in which the patients continue to remain well and free of symptoms for years subsequent to operation.

Local excision of a stricture or tumor of the duct has been followed in some instances by persistently good results over a period of years. The operation of the establishment of an external biliary fistula and its transplantation into the stomach or duodenum continues to afford hope to patients with complete stricture of the extrahepatic ducts for whom no other type of anastomosis is possible.

If I may comment on Doctor Ivy's interesting presentation, I should like to say that clinical observations carried out at the Mayo Foundation have demonstrated the presence clinically of this sphincter mechanism at the lower end of the common duct.

It consists of an operation upon the common duct, in which, when the patient has pain, it can be controlled by as small a dose as one-sixth of a grain of morphine, there occurring an increase in pain from the use of water. When the pain is experienced, the pressure can be recorded, and interestingly enough, when one-hundredth of a grain of nitroglycerin is given under the tongue, the pain subsides. Studying it further, they have used, in the common duct, an opaque substance, brominol, and administering morphine, in those patients who have pain after a cholecystectomy and who have had also an exploration of the common duct, and they have found that when the pressure within the duct increases one can see the sphincter contract and the intra- and extrahepatic ducts fill with this opaque media. Amyl nitrite inhaled will give immediate relief not only of the pain but also of the intrahepatic duct pressure. These procedures have been employed in several patients who have continued to have colic subsequent to operations on the gallbladder, with immediate relief of pain.

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Dr. Roscoe R. Graham (Toronto, Ont.).—Doctor Eliot's work has stimulated us all, and given us great help. We have had two cases from which we have learned lessons, which I would like to present to you. In the first case I was responsible for the subsequent complications. The patient was operated upon through a transverse incision. A very simple operation was rendered difficult because of the angle of approach. However, it was accomplished, apparently satisfactorily. We still belong to the group of surgeons

Fig. 1.—Injection of the sinus, showing the puddling of bile about the biliary ducts.

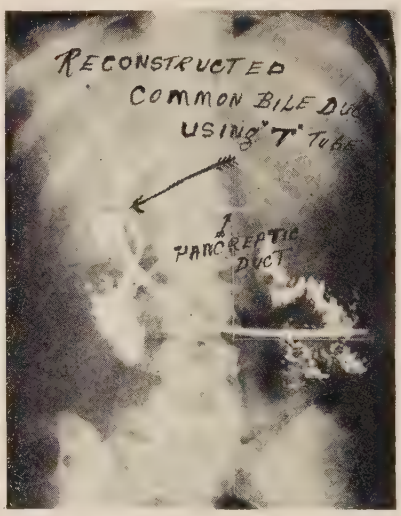


Fig. 2.—Showing the restoration of continuity of the biliary ducts and outlining the pancreatic duct.

who leave a drain in following the removal of the gallbladder, which is taken out on the third or fourth day. This was done in this case. Convalescence was uneventful for 48 hours. Then she began to complain of abdominal pain, had violent chills, and developed a mild jaundice accompanied by severe pain in the right shoulder: no drainage from the tube. Removal of the tube and investigation of the wound at the end of another 24 hours resulted in the evacuation of a large amount of bile. This temporarily relieved the situation, and she was allowed to return home. Shortly afterwards, however, there was a recurrence of pain, chills and fever, and she was admitted to hospital, intensely jaundiced, with frequent severe chills. Injection of the sinus (Fig. 1) showed accessory pockets and a puddling of the retained bile along the lateral margin of the liver, and a stricture of the common bile duct.

This gave us much food for thought, because I believe we were in error, and responsible for this situation, for two reasons: First: our approach was inadequate; second: our drainage was inadequate. We have since abandoned the transverse incision, but we still use drainage. However, the drainage tube

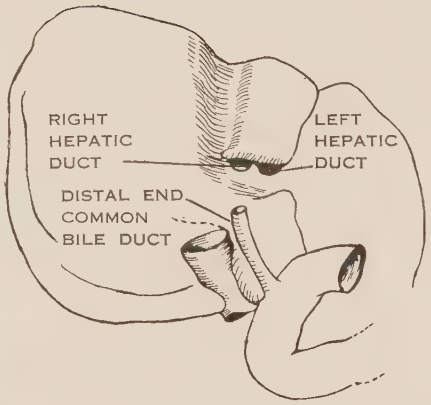


FIG. 3.—Showing the right and left hepatic ducts and the lower end of the common duct.

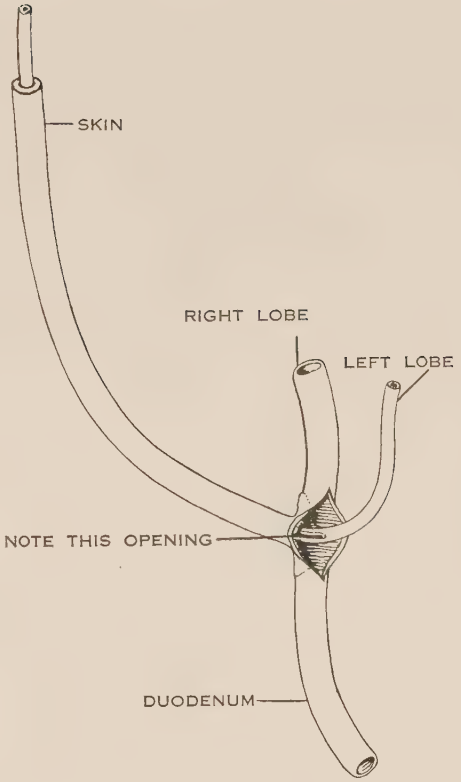


FIG. 4.—Showing the modification of the T tube to meet this problem.

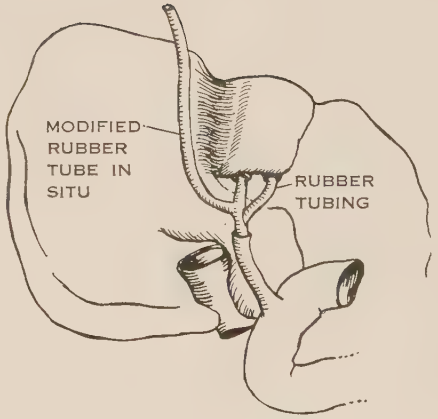


FIG. 5.—Showing the method by which the modified tube was placed in the ducts.

which is inserted has a segment of the circumference excised throughout its entire length, so that if we have the misfortune to have a leak from the cystic duct stump, it will find its way out along this channel before producing, as in this case, stenosis of the duct, the result of periductal edema from retained bile.

Figure 2 shows the outline of the biliary ducts and the pancreatic duct following the injection of lipiodol postoperatively. Her operation was completed March 21, 1934, and the patient, whom I have seen recently, is at the present time perfectly well, with no return of pain.

We were in a quandary following the taking of this picture as to the length of time that the T tube should be left in position. We have had, in the past, unhappy experiences following the too early removal of the T tube, due to a recurrence of the stenosis. Our problem in this particular case was solved for us, because, due to an accident, the tube was dislodged at the end of 15 months.

The second case, which I had the opportunity of seeing and operating upon with Dr. Harold J. Couch, presented a difficult problem. Six months previously she had her gallbladder removed. She remained well for about four months, when she began to have a return of colic, accompanied by chills and fever. Investigation led to the diagnosis of stenosis of the common bile duct.

At operation the situation as represented in Figure 3 was found, namely, two holes in the liver, which we were able to identify as the lower end of the right and left hepatic ducts, and a gap of about three-quarters of an inch between that and the isolated lower segment of the common duct.

To meet this situation we modified the T tube as shown in Figure 4, by passing a smaller tube through the long end of the T tube, bringing it out in the middle of the T. At this point it is important to have an opening cut in the smaller tube so that bile can find its way into the lumen of the second tube. The tube was placed, a limb in each right and left hepatic duct, and the third in the common duct, as indicated in Figure 5. Then, by mobilization of the duodenum, it was possible to approximate the common duct to the ends of the hepatic ducts. This operation was completed in July, 1933. The tube was removed at the end of six months, after injection of the ducts with lipiodol demonstrated their patency. This patient is, at the present time, well, and has had no recurrence of her symptoms.

Dr. J. Shelton Horsley (Richmond, Va.).—We are all very much indebted to Doctor Eliot for his splendid work on surgery of the common duct, not only as given in this paper but in previous communications.

A good many years ago I attempted experimentally to reconstruct the common duct and used a segment of vein. The use of a vein was not original with me; I do not know where I got the idea, but I worked out a rather complicated technic. Most of the dogs died, but those that recovered I kept, and after a few months they all developed jaundice and died or were killed. I think the one that lived longest lived eight or nine months. The postmortem on that dog, as well as on the others, showed that the segment of transplanted vein (which was inverted and looked like ideal tissue from a mechanical standpoint, thin, and easily nourished) was invariably infiltrated with leukocytes, eventually contracted and formed a complete stricture.

In other words, in reconstructing the common duct, it is necessary to know biology. Tissue that is transplanted must be more or less accustomed to the environment, and to the secretions. The vein was unaccustomed to, and irritated by, bile, and deep leukocytic infiltration was followed by stricture.

If there can be a direct anastomosis of the duct, that is very good, but if that cannot be accomplished, anastomosis to the duodenum is the second best choice.

We also have to remember that hepatitis is a thing that is more frequent than we suspect. The work of Evarts Graham many years ago showed it as a very common occurrence with even a slight inflammation of the gallbladder.

The happy results that Doctor Eliot has found in anastomosis of the common duct to the duodenum have not been entirely my experience. I recall one case in which it was necessary to do that because the gallbladder had previously been removed. I did a lateral anastomosis between the common duct and the duodenum. The patient made a fairly satisfactory immediate recovery, had more symptoms a few months later, and on roentgenologic examination with barium meal, an almost perfect pattern of the common and hepatic ducts could be seen. I wanted to disconnect the anastomosis, but he would not let me do it and he died a few months later with liver symptoms. Possibly an end-to-side union would have been better.

We owe much of the improvement in modern surgery, especially in the gastro-enteric tract, to the physiologist. It has been through disregarding many of the principles laid down by the physiologist, that we have gotten into trouble. Almost any anastomosis between the gallbladder and the common duct that is followed by hepatitis puts us in a hole, but it is often a condition, not a theory, that confronts us. We do not want to do it, but we have a patient who is certainly going to die unless we give some relief by drainage, and may die if we do. Anastomosis of the gallbladder to the duodenum would seem from a physiologic standpoint to be the most satisfactory of any of these operations, but even that undoubtedly is sometimes followed by unfortunate results. I think, however, that hepatitis in man seems to be better tolerated and does not have quite such an inevitable and unfortunate result as in a dog. Of course I cannot prove that, but I know some cases seem to continue for a long time with hepatitis, living pretty comfortably. We have so little idea of the actual functioning of the diseased liver, and the tests are so unsatisfactory, that not infrequently we have to make use of these procedures, even though they may be undesirable from a physiologic standpoint.

Dr. John Douglas (New York, N. Y.).—Just ten years ago I read a paper on stricture of the bile duct, before this society, and reported 12 cases. At that time, the follow-up would seem to indicate that the results with anastomosis between the ends of the duct when possible were the most favorable. Two of these cases of my own were cases in which anastomosis had been made between the divided ends of the common duct, in one instance of the common duct itself, and in the other the junction of the hepatic and common ducts. I have been able to follow up both of those cases in the last few months, at Doctor Eliot's request, and both were still well, one 11 and the other 14 years after operation.

A point which is of considerable interest to me in those cases is that two or three years after the operation both patients had decreasing attacks of cholangeitis. They had jaundice, chills, and rises of temperature. Doctor Ivy has told us that this is more apt to occur in the case where the sphincter of Oddie has been destroyed. Probably this is so, but both of these patients still had their sphincter of Oddie, and they both had jaundice with temperature.

We know that in cases of stricture of the common duct where there is jaundice, and in many cases where there are stones in the common duct, sometimes we will find a lot of muddy material in the common duct and sometimes almost cast like material, such as Doctor Ivy has spoken of.

Doctor Whipple has said if we could only find some way to prevent this cholangeitis he would have more confidence in the possibility of these various operations. I do not know any way to prevent the cholangeitis. However, these cases of mine seemed to get over the attacks more rapidly when given bile salts. There is still a question, of course, as to the effects on the flow of bile, but the presence of bile salts in bile, as is known, will keep the cholesterol in better solution, and the absence of bile salts will encourage its precipitation. It is a simple thing, but it seems in those two cases which I have followed over 10 and 14 years, respectively, when they got the bile salts they recovered from the attacks of cholangeitis more rapidly than when they did not receive it; and whereas they got the attacks of cholangeitis over two or three years or more, within the last two years something has happened which is now preventing their attacks of cholangeitis.

Dr. Ellsworth Eliot, Jr. (New York), closing: I have mentioned the administration of bile salts as a prophylactic postoperative measure for subsequent attacks of cholangeitis. There are, in addition, two other forms of treatment mentioned for this purpose: (1) the injection into the duodenum of a solution of magnesium sulphate, and (2) the administration of secretin. Irrespective of the type of suture or anastomosis, the postoperative attacks of cholangeitis are usually transitory, although, exceptionally, they may terminate in an ascending infection with invasion of the liver parenchyma.

The T tube has enjoyed great vogue, but Horgan, of Washington, has substituted an L shaped tube which may be readily withdrawn when desired without danger of rupture of the line of suture of the duct itself. Selim McArthur has also devised a substitute for the T tube quite similar to the L shaped tube but differing from it by being supplemented by the introduction of a second smaller tube which passes downward into the duodenum and through which medication may be introduced. Both of these varieties of duct drainage would seem to diminish the probability of rupture of the line of suture and subsequent recurrence of stricture.

Brandt, in 1912, reported five cases operated upon by Wilms in which the immediate result was highly satisfactory (duct reconstruction with buried tube). In two cases, 18 months, and in the others, a much shorter period has elapsed since he published his observations. Through the kindness of Professor Kerschner of Heidelberg, I am able to report that four of these patients died, two, two, 11 and 17 years, respectively, after operation, the first mentioned of gallstone disease. The cause of death of the next three could not be ascertained, while the fifth patient could not be traced. The unfortunate outcome of these cases may account in part for the unpopularity of the Wilms operation abroad.

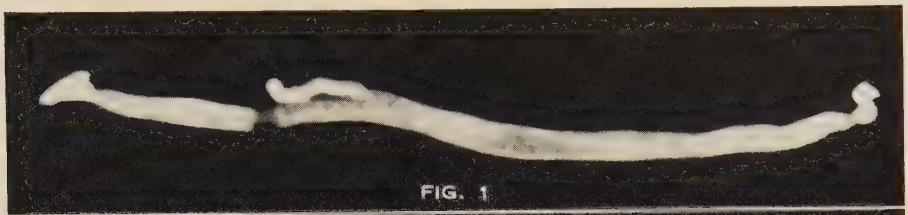
Other surgeons besides Doctor Lahey have used the Mikulicz procedure, namely, widening the duct at the point of repair. I am under the impression that this procedure was used in several instances by the late Doctor Judd of Rochester and was followed by a recurrence of the stricture.

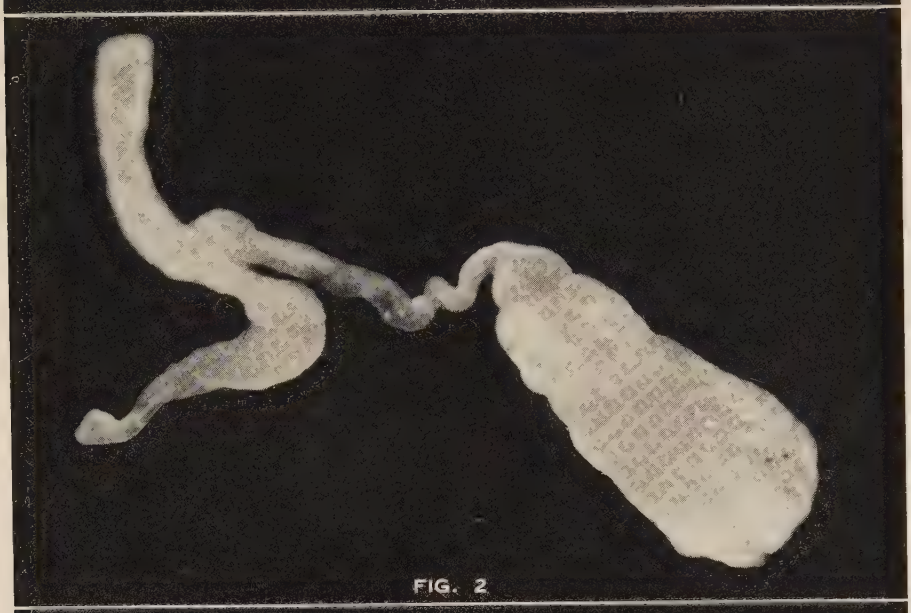
Regarding Doctor Horsley's remarks, I am told by Dr. Dean Lewis that experimental substitution of the wall of a vein or other fascial structure for the excised segment of a duct terminated in failure. Furthermore, plastic operations in which portions of adjacent hollow viscera were utilized for a similar purpose were generally unsuccessful and have been discontinued.

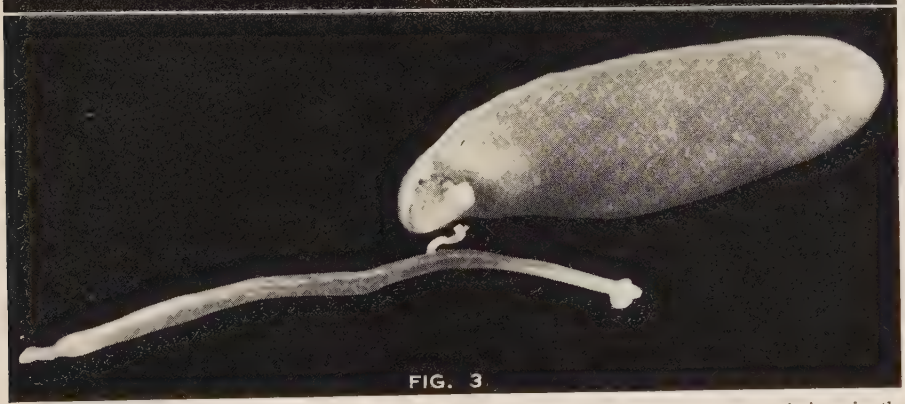
Failure of the bile to properly drain after cholecystectomy, to which Doctor Graham has referred, occasionally occurs. Two instances observed by colleagues in which such an accumulation occurred in the lesser peritoneal cavity, resulted fatally. Doctor Lahey, recognizing the possibility of such an

unfortunate postoperative complication, regularly introduces a suitable drain through the foramen of Winslow.

The retention of the drainage tube is, at times, difficult to control. In operations for stricture of the hepatic duct, retention is favored by the intro-







FIGS. 1, 2 and 3.—Paraffin casts of gallbladders, cystic and common ducts showing variations in the caliber of corresponding parts.

duction of a Y shaped tube into the right and left hepatic ducts, the vertical part of the Y extending into the duct below. Furthermore, a number of operations have been performed, especially abroad, in which retention of the

tube has been insured by bringing it out through a Witzel opening in the wall of the duodenum or stomach into which the tube has been introduced, which emerges with the drain into the dressings, where it is firmly secured to the abdominal wall. Its withdrawal at any time, according to the judgment of the surgeon, can be effected without disturbing the repair of the duct wall.

In the Hildebrandt operation, the jejunum is divided a little below the jejunoduodenal junction, and intestinal continuity is reestablished by lateral anastomosis of the oral jejunal orifice about 10 cm. below the point of division. The distal jejunal orifice is then brought out of the abdominal incision and inserted through a tunnel behind the rectus sheath to the orifice of the biliary fistula with which it is anastomosed. This operation has been performed three times. In one case, the patient was alive and well ten years after the procedure; in the second, death occurred from ascending aseptic cholangeitis, and in the third, it was a failure, another operation being subsequently performed. A modification of this procedure has been adopted by Dobrotworsky of Leningrad. The chief difference is that no anastomosis is attempted, but in its place the biliary fistula and the distal orifice of the jejunum are connected by a glass tube which can be removed, cleaned and reinserted at will. Through the glass tube in situ, the bile can be seen passing into the intestine. Several instances of this operation are reported by the author, one patient being alive and well 12 years later.

I have always thought it possible that the caliber of the normal hepatic and common ducts might not be uniform and might be subject to variations in different portions. With the cooperation of Doctor Helfrick, intern in the Knickerbocker Hospital, cadavers of adults, who had died violent deaths, were selected for investigation. After ligation of the hepatic ducts above and at the ampulla below, the gallbladder and cystic duct were injected with paraffin. After this had hardened, the enveloping soft tissues were dissolved by hydrochloric acid which left a cast of the interior of the biliary ducts and gallbladder. Each specimen treated in this way showed variations in the caliber of the duct. The caliber of corresponding parts of the duct showed considerable variations. These variations were most pronounced in the specimens in which the gallbladders were still attached (Figs. 1, 2, and 3).

It is not inconceivable that such variations in the normal ducts, especially at the junction of the cystic and hepatic ducts and at the ampulla, may predispose to the development of stricture in the presence of a calculus cholangeitis.

CHOLECYSTODUODENOSTOMY COMBINED WITH PYLORIC EXCLUSION

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THE increasing number of papers dealing with operations for the anastomosis of the biliary passages with the digestive tract indicates the great interest which this subject attracts from the clinical and the experimental surgeon. For comprehensive surveys of the literature in this field, the reader is referred to the papers of Gentile,[7] in English, Berard and Mallet-Guy,[2] in French, and Bernhard[3] and Hutter,[9] in German.

In animal experiments, a dilatation of the bile ducts and an ascending infection leading to hepatitis and cirrhosis almost invariably follow anastomosis of the bile ducts with the gastro-intestinal tract. The prominence of the hepatic lesions depends to some extent upon the site of the anastomosis, being least severe in the presence of cholecystogastrostomy and cholecystoduodenostomy and increasing in degree as the communication is made lower in the intestinal canal.

Clinical findings, on the other hand, have not been uniform. Most surgeons have considered that, in man, an ascending infection of the biliary tract rarely follows these anastomoses, and, on this basis, some have recommended extension of the indications for such operations.

Several hypotheses have been advanced to explain this supposed difference in reaction between man and dogs, among which are: anatomic differences, greater bacterial content in the digestive canal of the dog, and possibly a greater resistance to infection possessed by the human liver.

When, however, one closely examines the facts, it appears that the conception, that an ascending infection of the biliary tract occurs less frequently in man than in dogs, is not well founded.

While it is true that, in many cases, the anastomosis of the biliary tract to the digestive canal in man is not followed by the development of unfavorable symptoms, this is equally true in animals. Most dogs are apparently healthy after the operation, and cholangeitis and hepatitis, even with liver abscesses, may not be discovered until autopsy is performed. Statements that ascending cholangeitis rarely develops in man are usually based upon clinical impressions rather than upon evidence from postmortem findings. The more thoroughly the patients are examined after the operation, the more frequently are signs of infection discovered. For example, Bernhard[3] reports an inci-

dence of signs of infection in from 10 to 20 per cent of patients, and Hutter[9] in 11 per cent.

A review of the reported cases of biliary tract anastomosis in man reveals that when the liver has been examined at autopsy signs of cholangeitis and hepatitis have been found in most instances.

We have found 20 cases in which definite evidence of infection was found at autopsy: one case with cholecystoduodenostomy,[27] nine cases with cholecystogastrostomy,[9, 12, 18, 20, 21, 23, 28] one case with choledochoduodenostomy,[4] two cases with cholecystojejunostomy and entero-anastomosis,[11] six cases in which the situation of the anastomosis was not noted,[1, 6, 14] and one case of Judd's cited by Gatewood and Lawton,[6] in which a patient with cholecystogastrostomy who died of intercurrent disease exhibited hepatitis with abscesses, in spite of the fact that there had been no indication of liver damage before death.

In addition to four cases developing cholangeitis following anastomosis of the gallbladder to the gastro-intestinal tract, Leven[14] reported one case in which there was improvement of a previously existing cholangeitis after such a procedure. He also reported eight cases which he considered to be normal except for changes which regularly follow biliary obstruction. Those changes, however, included an increase in the amount of portal connective tissue and a periportal round cell infiltration. Since similar changes may be produced by an ascending infection, it is impossible to classify such cases accurately.

We have found only two cases in which the presence of hepatic infection was definitely excluded at autopsy: one case of cholecystogastrostomy[13] and one case of choledochoduodenostomy.[8]

In interpreting these figures, however, one must remember that some of the infected cases presented evidence of liver injury before the anastomotic operation, and also that cases without cholangeitis probably would be less likely to come to autopsy than cases with hepatic damage. Furthermore, as Leven has pointed out, simple biliary obstruction may lead to changes in the liver which present a confusing picture.

Several procedures have been suggested to lessen the danger of infection. Most of these are based on the principle of diverting the chyme from the anastomosis in order to prevent the entrance of infected material from the alimentary canal into the biliary tract. Krause[27] established an anastomosis between the gallbladder and the jejunum, and performed entero-anastomosis between the afferent and efferent jejunal loops. Monprofit[19] suggested cholecystojejunostomy in the form of a Y, and Marchetti[17] reported good results after experimenting with this method.

Strauss,[25] following the same principle, combined gastro-enterostomy with choledochoduodenostomy in 22 human cases, and reported that he had thus avoided cholangeitis. In one of his cases, symptoms of cholangeitis which had developed following choledochoduodenostomy disappeared after secondary gastro-enterostomy. Mallet-Guy[16] reported a similar case. On the

other hand, Whipple[28] and Hutter[9] reported three cases in which cholangeitis was found at autopsy in spite of the fact that cholecystogastrostomy and gastro-enterostomy had been performed simultaneously.

It is clear that there has been a need for experimental control of the results obtained by procedures such as that advocated by Strauss.[25] We have undertaken such a controlled study, and Gentile,[7] at about the same time, carried out a somewhat similar series of experiments, the results of which he has recently reported.

Method.—In a series of 14 dogs, under morphine and ether anesthesia, and using aseptic surgical technic, we anastomosed the gallbladder to the duodenum, interrupted the continuity of the common bile duct (by ligature

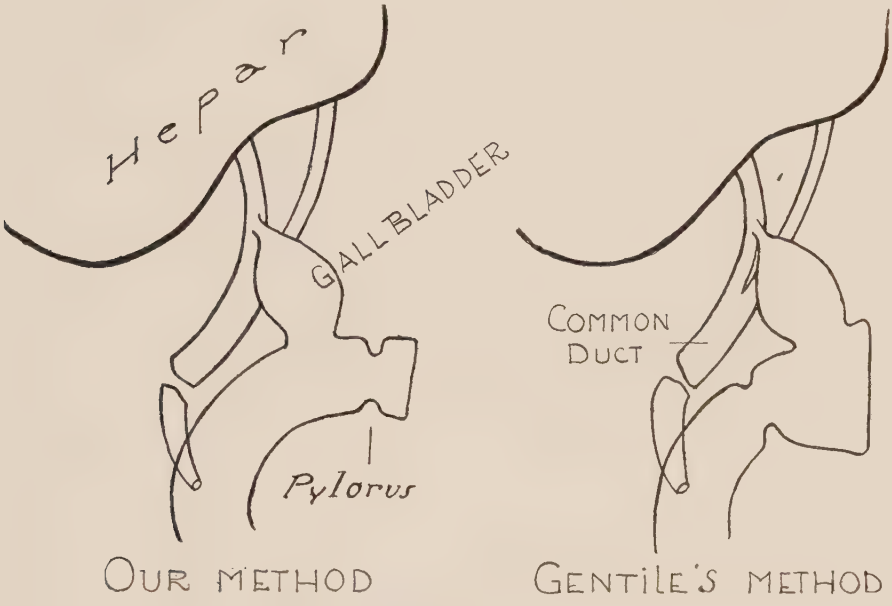

Fig. 1.—Diagram of our operation and that of Gentile.

in one-third, and by division in two-thirds of the cases), excluded the pylorus, and performed gastrojejunostomy in a single stage operation (Fig. 1). At the same time we excised small pieces of the liver and the pancreas for microscopic examination, to serve as controls for comparison with tissue taken at a later date. We added pyloric exclusion to the gastro-enterostomy in order to be more certain of diverting the chyme from the duodenum.

Liver function tests were carried out on all dogs both before and at regular intervals after operation. The tests used were: The diazo test of van den Bergh, the icterus index of Meulengracht, the serum pigment test of Ernest and Forster, the bromsulphalein test of Rosenthal and White, and the blood lipase test of Cherry and Crandall. At the same time the cage urine was collected and tested for bilirubin by the method of Hooper and Whipple.

The dogs were sacrificed with ether at intervals of two to ten months

TABLE I

POSTOPERATIVE CHANGES

Dog No.	Post-mortem Time After Operation	Circum. of Common Duct Mm.	Circum. of Anasto-mosis, G. B. to Duo-denum Mm.	Grossly Contaminated		B. Coli, Number per 0.1 cc.		Liver Histology		Jejunal Ulcer	Weight Changes % + gain − loss	Comments
				G. B.	Ducts	Periph-erally	Cen-trally	Peribil-iary Infiltra-tion	Infiltra-tion Fat			
1	2 mo.	18	14	+	+	—	—	++	+	0	−	
2	2.3 mo.	20	8	0	0	5	10,000	+	++	0	−17	
3*	3 mo.	15	20	+	+	—	—	+	−	0	−7	Purulent cholangeitis
4	3.4 mo.	10	28	+	+	21	25	+	−	0	−13	
5*	4.4 mo.	15	15	+	+	—	—	+	+	+	−17	Perforated ulcer
6	4.5 mo.	20	10	+	+	1	1,000	++	−	+	−30	
7	4.7 mo.	23	10	0	0	300	10,000	+++	−	0	−35	
8*	5.3 mo.	24	16	+	+	—	—	++	−	+	−25	Perforated ulcer
9	7.8 mo.	12	15	0	0	800	200	+	−	0	−18	Duct reestablished Duct reestablished
10	8 mo.	12	10	—	—	0	15	+++	−	+	−44	Concretions
11	8.5 mo.	13	10	+	+	0	1	++	++	0	0	
12	8.5 mo.	10	14	+	+	1	200	+	++	0	+11	
13*	10.7 mo.	16	12	+	0	—	—	+	++	+	−37	
14	10.7 mo.	16	10	+	+	0	0	++	++	0	−10	

* Died.

after operation. For two days prior to sacrifice, finely divided charcoal was mixed with the food so that the path of the ingested material could be easily followed. At the time of the autopsy, small sections were taken with aseptic technic from the center and periphery of the liver and from the spleen (control) for quantitative bacterial study. The biliary tract was examined carefully for gross contamination, and sections of the liver and pancreas were taken for microscopic examination.

RESULTS.—The essential results are recorded briefly in Table I.

Four of the 14 dogs died; one of purulent cholangeitis and three from

FIG. 2.—Calculi which developed in the biliary tract of Dog 10. The size of the stones may be judged by comparison with the inch rule scale at the side.

complications of jejunal ulcers. The secondary mortality, therefore was 28 per cent, of which only 7 per cent was due directly to liver infection.

In spite of an adequate diet, 12 of the 14 dogs lost weight, and this was most marked in the animals which developed jejunal ulcer.

In five animals (35 per cent), a chronic ulcer developed in the wall of the jejunum opposite to the gastro-enterostomy stoma.

No impairment of liver function could be detected by the use of the several tests enumerated above. Two animals, however, developed a bilirubinuria a short time before they died, and this was interpreted as being due to liver damage.

Autopsy revealed that the continuity of the ductus choledochus had become

reestablished in two cases. In other respects these animals did not differ from the rest of the group. The gallbladder anastomosis was patent in every case, and varied in circumference from eight to 28 millimeters.

Dilatation of the bile ducts was constant. The common duct measured from 10 to 23 millimeters in circumference above the point of its division, whereas normally it measures less than seven millimeters.

There was macroscopic contamination of the biliary passages in ten cases. In six of eight dogs which had been fed charcoal, grains of carbon were found in the biliary tract. In one animal round worms were found in the gallbladder and in the right hepatic duct.

One animal (dog 10) developed numerous calculi in the gallbladder and

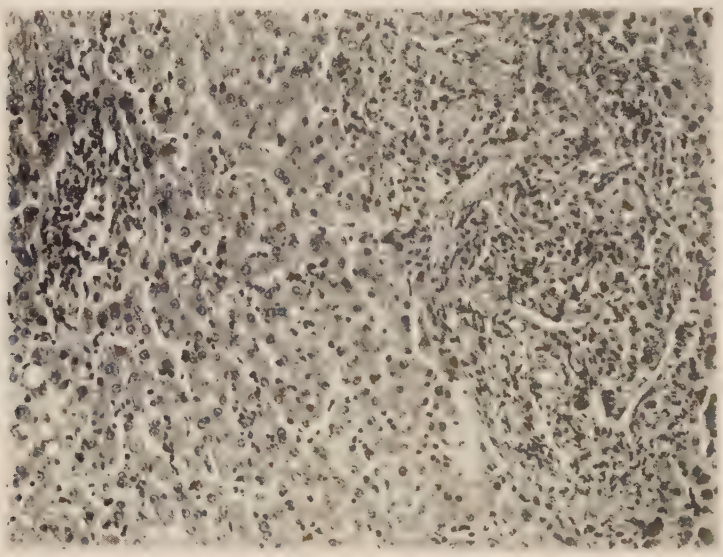

FIG. 3.—Photomicrograph of liver showing severe hepatitis occurring after cholecystoduodenostomy and pyloric exclusion.

ducts (Fig. 2). The concretions in the intrahepatic ducts were golden-brown and tubular. Similar concretions were present in the gallbladder, which also contained larger, irregular, dark-brown, pigment stones. *B. mucosum-capsulatum* were isolated from the liver of this animal.

There was no gross pathology evident in the liver in half of the cases. In one case (dog 10) the liver was hobnailed and cirrhotic, and calculi were present in the bile ducts. In another instance (dog 3) there was purulent material in the intrahepatic ducts. In the remaining animals, the only change was the appearance of areas of fatty infiltration which had not been present before the operation (Gage[5]).

Microscopic examination of sections taken at autopsy, compared with controls taken at the time of operation, revealed the pancreas to be normal in all cases. Hepatitis, however, was present in every instance. There was peribiliary infiltration with round and plasma cells, and often with large num-

bers of polymorphonuclear leukocytes (Fig. 3). There was present a fibrosis of varying degree. In half of the dogs the liver cells showed fatty infiltration in some areas.

Quantitative bacteriologic studies were carried out by Dr. W. J. Nungester, of the Department of Bacteriology. In seven of the nine dogs thus studied, a lactose-fermenting, gram-negative bacillus (presumably B. coli) was present in the liver, sometimes in large numbers, and especially in the central portion.

Discussion.—It is evident that none of the tests of liver function which we have used was sufficiently sensitive to detect even severe degrees of ascending hepatic infection. This emphasizes the large factor of safety in the liver, which accounts for the satisfactory general condition of patients even in such serious cases as that of Judd.

Dilatation of the bile ducts constantly follows anastomosis of the biliary tract to the digestive canal. In normal individuals, closure of the sphincter of Oddi shuts off the bile passages from the duodenum during periods when the pressure within the intestine is high.[15, 22] Removal of this protection permits transmission of pressure into the ducts from the duodenum, and it appears that this transmitted pressure may explain the dilatation. It is probable that infection contributes to the process by weakening the walls of the bile passages.

It would also appear from the results which follow artificial connections between the biliary passages and the gastro-intestinal tract, that the sphincter of Oddi, or the normal choledochoduodenal mechanism, serves not only the well established function of rendering it possible for the gallbladder to fill, but also of preventing or reducing the likelihood of ascending infection of the biliary passages. It would further appear that in the reconstruction of the bile ducts it is preferable to preserve the choledochoduodenal mechanism, when possible.

Cholecystoduodenostomy combined with exclusion of the pylorus, in this study, has invariably led to an ascending infection of the liver. Such an operation, therefore, does not appear to have any definite advantage over simple cholecystoduodenostomy. This conclusion draws further support from Whipple's[28] and Hutter's[9] three cases of cholecystogastrostomy in which simultaneous gastro-enterostomy failed to prevent the development of fatal cholangeitis. We entertain serious doubts regarding whether any surgical procedure may be devised which will substitute for the choledochoduodenal mechanism to such an extent as to reduce markedly the tendency toward the occurrence of cholangeitis.

Gentile[7] subjected his dogs to gastric division at some distance from the pylorus, and a Pólya-Balfour anastomosis. Later he established a communication between the gallbladder and the relatively bacteria free, distal, blind end of the stomach. He considered that he had produced by this method conditions comparable to those existing after cholecystogastrostomy in man. His animals, however, would seem to be somewhat more favorably situated

because of the diversion of the chyme, and it would be necessary to divide the stomach in man also, if one desired to duplicate the conditions of his experiment. He reports that the hepatitis which developed in his 22 dogs after cholecystogastrostomy, according to his method, was not more pronounced than after simple exclusion of the pylorus. A survey of his tables, however, shows one case of severe ascending infection with liver abscess and fatal termination, and several cases which showed a marked hepatitis at autopsy. Not even with Gentile's complicated method, therefore, is it possible to avoid

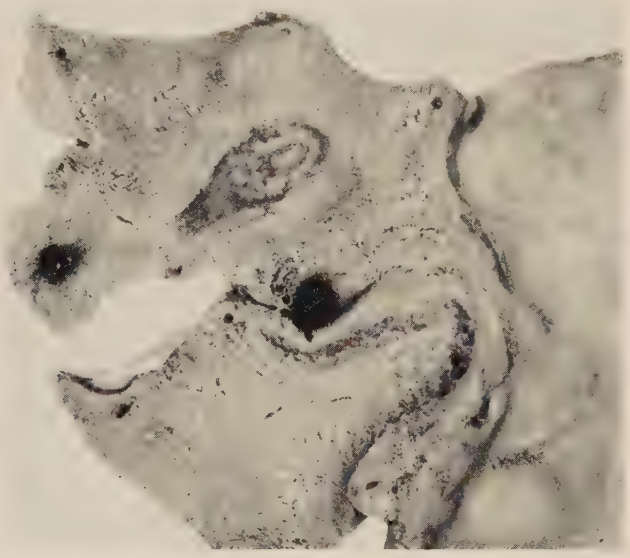

FIG. 4.—Photograph showing jejunal ulcer which developed after cholecystoduodenostomy and gastro-enterostomy with pyloric exclusion. This was the smallest of the five ulcers which developed. Hemorrhage from the ulcer caused the death of the animal.

the danger of ascending infection. Moreover, the secondary mortality in our material, as well as in Gentile's, is considerably increased by a high percentage of jejunal ulcer (Fig. 4). This risk is also present in man (Walzel)[26].

The association of ulcer with disturbances of the liver or its secretions has been noted in many instances.[10, 24] The significance of this relationship is not clear, but the suggestion recently offered by Schnitker and Hass,[24] that the peptic ulcer may arise as a result of some deficiency, is interesting.

CONCLUSIONS

(1) Attempts to prevent ascending infection following bile duct anastomosis in dogs, by diverting the chyme so that it does not pass the anastomosis, have been unsuccessful, both in our investigations and those of other experimental surgeons.

(2) To effectively divert the chyme, it is necessary to perform pyloric exclusion in addition to gastro-enterostomy. Even after pyloric exclusion, some of the ingested material makes its way back into the duodenal loop.

Since this procedure undoubtedly adds to the operative risk, in view of our findings it does not seem to be justified.

(3) It appears that ascending infection usually results following bile duct anastomosis in man, as well as in animals. Since the factor of safety in the liver is so large, this infection rarely gives rise to clinical symptoms. Occasionally, however, a fatal infection may ensue, especially if stasis of bile occurs. The development of peptic ulcer in some cases constitutes an added danger. For these reasons it would seem unwise to extend the present indications for this operation.

(4) In the presence of an irremovable obstruction in the terminal portion of the common bile duct, simple anastomosis of the gallbladder to the stomach or duodenum is a satisfactory operation. More complicated procedures add to the operative risk without presenting any definite advantages.

(5) A normally functioning sphincter of Oddi, or choledochoduodenal mechanism, plays an important rôle in the prevention of cholangeitis and dilatation of the bile ducts.

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Discussion.—Dr. Andrew C. Ivy (Chicago, Ill.) in his remarks closing the discussion stated that one of the functions of the sphincter of Oddi which has been established is that it makes possible the filling of the gallbladder. And I think in the course of time the sphincter of Oddi will be given a second function, namely, that when it is functioning normally it prevents ascending infection and anatomic changes from occurring in the ducts.

The contention that sometimes even though you leave the sphincter of Oddi in place and reconstruct the duct and get cholangeitis, the sphincter does not have anything to do with it, does not necessarily follow logically, because we have instances of cholangeitis in human begins who have never had their biliary tract operated upon. I should say that we should attempt to preserve the sphincter mechanism whenever possible.

In so far as the development of various complicated and involved operations for anastomosing the biliary passages with the gastro-intestinal tract is concerned, I think the possibilities have just about been exhausted, at least in the dog. In so far as studies on the reconstruction of the ducts, and the incidence of cholangeitis after such procedures, are concerned, I do not think the experimental possibilities have been exhausted. I suspect that the clinical possibilities have been exhausted, but should be further analyzed.

In regard to Doctor Douglas' point about the bile salts, I do not think there is any question concerning the point that bile salts when given intravenously or given by mouth stimulate the formation of an increased volume of bile. We have biliary fistula animals in our laboratory now, in which if we drain the bile to the outside, the bile output of the liver decreases; if we divert the bile back into the intestine, the bile output on the same diet increases.

In that connection and in regard to one of Doctor Eliot's remarks, we are now using, with excellent success, Doctor McArthur's procedure in which one catheter is placed into the common duct pointing towards the liver and another smaller catheter is passed through the ampulla into the duodenum. In this way we may administer anything we desire via the intestine. I personally believe that this method has a number of advantages over a T tube.

In doing this work I have in mind a fundamental problem which concerns the surgeon. I am interested in more than the functional activity of the sphincter of Oddi. I should like to discover some way of preventing ascending cholangeitis. Not having had the desired success by surgical methods, I have had a man working for about 18 months on biliary antiseptics, with the hope that when we suspect that a cholangeitis might occur, we might have some biliary antiseptic to give which would reduce the likelihood of a cholangeitis. We have used many of the so called antiseptic dyes and drugs. Surprisingly, the best "antiseptic" we have found to date is salyrgan.

TWENTY-FIVE YEARS' EXPERIENCE IN THE TREATMENT OF PERITONITIS

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IN AN introductory paper on septic peritonitis read before the 1935 meeting of the Northern Surgical Society, Bohmansson[1] failed to submit any statistics, because, as he stated, "we meet with such difficulties in our statistical elaboration of our peritonitis cases that we are unable to understand each other's language and figures." It is surprising to hear such words from a clinician. A careful study, even if burdened with figures, of extensive, uniformly treated, clinical material, with a critical estimation of cases, has always been and will always remain the rock on which we must build up our surgical knowledge. Should we permit ourselves to abandon the method because of its difficulty, we are certain to fall back on mere speculation, personal bias and casuistic chatter. If we really no longer "understand each other's language," it is high time we tried to find out the reason. We must come to an understanding in the question and I am sure we will do so before long.

As a young doctor I was instructed by Lennander, my old teacher, to make a critical study of his material of acute appendiceal peritonitis. This task taught me how necessary it is for a correct view of our clinical experiences to examine case records in detail and without prejudice in the bright, but often unpleasant, light of criticism. The experimental surgeon can, as a rule, create the pathologic conditions he wishes to study from any given starting point and can study critically the methods of treatment he considers useful. This is impossible for the clinician. He does not select his cases; he gets them for observation and treatment. It is only a careful study of his collected material over the course of a long period of time that enables him to obtain a bird's-eye view, minimizing chance coincidence and clarifying the broad aspects of the problem.

My own material comprises all cases observed as senior surgeon in Umeå from 1910 to 1921, in Stocksund from 1922 to 1927, and at the II[nd] Surgical Department of Sabbatsberg Hospital, Stockholm, from 1928 to 1934. The Umeå hospital had at that time only one senior doctor, who treated both medical and surgical cases. At the Stocksund, as well as at the Sabbatsberg hospitals, there was a separate surgical department. At Stocksund the department also received gynecologic cases. One reason we do not understand each other in our discussions on peritonitis is that we still today speak of it as a uniform disease. We entirely forget that often the only common feature in our peritonitis cases is that the disease takes its course within the human abdominal cavity, and naturally certain com-

mon or similar reactions take place between the organism and the peritoneum. Yet the fact is that the nature and degree of the infection, as well as the different locations and structures of the abdominal organs and their various reactions to external and internal injuries, are of such manifold character that we really must consider a vast number of different forms of peritonitis, forms which frequently have little more in common than the name and each of which demands its specific treatment. As a proof of the validity of this statement I may mention Linzenmeier's[2] classification of pelvic peritonitis in the female which includes no less than ten well differentiated forms.

Before we enter a general discussion of acute peritonitis we must attempt to solve without confusion all disputable questions concerning each special form of peritonitis. It may be that at some time in the future we may attain this goal and I feel certain that then there will be no more peritonitis to discuss. If we study carefully and conscientiously the origin and development of every form of peritonitis, we shall more and more understand that the only proper treatment, as always, is prophylaxis. Our aim shall then be not to *treat* peritonitis but to *prevent* it. Is it presumptuous to dare to hope for this? I venture to think not. Nearly all patients with peritonitis complain of pain, generally of a very severe nature, and the sufferer knows that he is ill. Fortunately, in most cases of peritonitis there is an interval between the onset of the disease and the point at which it passes into an irreversible stage. This interval varies in length in different forms of peritonitis and it may be very short. We must make use of this interval by removing or shutting out the source of infection. It is true that some forms of peritonitis still exist in which it is difficult to eliminate the source of infection, *e.g.,* pancreatic peritonitis and the purulent form of idiopathic peritonitis, the genesis of which we still do not know or sufficiently understand, but fortunately these forms are quite rare.

Another reason why we often do not understand each other is that we have generally failed to realize, in considering the same form of peritonitis, the importance of the different stages of its development. We compare cases which present certain superficial similarities, but which often possess widely different fundamental characteristics. In this respect, the terms "localized" and "free diffuse" have created and still create a great deal of confusion in the field of appendiceal peritonitis. It is now generally accepted that the exudate in purulent appendiceal peritonitis is in its first stage a free and not a walled off seropurulent fluid. It is later, as the admixture of leukocytes becomes more plentiful, that thick pus develops and fibrin is precipitated, making up the substratum for encapsulation. As a rule, fetid disintegration and encapsulation begin only at the end or at the beginning of the third day after onset of the symptoms. Why an appendiceal peritonitis becomes walled off in one case, and in others tends to spread over a considerable part or the whole of the peritoneal cavity, we do not know in detail; but we have reason to assume that the chief rôles are played by the intensity of the infection,

the spread of destruction in the appendix, and the position of the appendix at the onset of disease. But perhaps most important of all is the bursting of peri-appendiceal abscesses through feeble fibrin walls with discharge of their contents into a previously noninfected abdominal cavity.

When, therefore, we operate upon a patient with appendiceal peritonitis on the first or second day after the onset of the disease we shall find, in the right iliac fossa, or in this fossa and the small pelvis, or in certain exceptional cases in the abdomen below the colon, or in other, also exceptional, cases in the right part of the abdomen toward and in the hypochondrium, a seropurulent or true purulent fluid, usually odorless and generally without any sign of encapsulation. To give a prognosis on the basis of clinical signs in this stage has proved impossible; it has, therefore, become a conviction, shared by all the world's surgeons, that at this stage operation should be carried out without delay. Should one postpone such an operation, one would find that in a certain number of cases the peritoneal exudate had become absorbed and that the appendix was on the point of organization after its destruction; in other cases a more or less walled off abscess would have formed around the appendix as a nucleus. On the third to fourth day in such cases we would find around the encapsulation another serous to slightly turbid exudate. This, too, however, would be reabsorbed, after which the peritoneal cavity around the encapsulation would resume a perfectly normal appearance. In a third category of cases, proceeding neither to reabsorption nor to encapsulation, one would find a peritonitis extending over varying parts of the abdominal cavity and presenting in its later stages the well known picture of appendiceal peritonitis at the necropsy table—a fetid fibrinopurulent peritonitis with a number of more or less completely walled off pockets of pus.

It will be clear from this description of the various grades and modes of development of appendiceal peritonitis that, as regards its character and prognosis, a purulent appendiceal peritonitis is most imperfectly described by the expressions "localized" or "free diffuse." With few exceptions the term "free diffuse" includes all those cases of purulent appendiceal peritonitis operated on within 48 hours after the onset of the disease, as well as those cases in which Nature's own healing powers have failed to absorb or encapsulate the infection and which make up the majority of all peritonitis cases having a fatal issue. It would seem impossible, even when operation is carried out within 48 hours, to decide with certainty along which of the three lines the individual case might have progressed had no operation been performed. It is true that in some cases an early encapsulation may be noticed, while in others the benign appearance of the exudate may lead one to assume a probable reabsorption or encapsulation. My experience, however, with this and later stages, has taught me the impossibility of determining the prognosis from the gross appearance of the peritonitis. Some cases that appear particularly benign proceed without fail to the postmortem table; others, of an extensive nature with an ugly exudate and greatly altered intestine, pro-

ceed as quietly as any other postoperative case. Thus in the great majority of cases presenting "free diffuse" appendiceal peritonitis at operation, particularly within 48 hours of the onset of illness, there is an intrinsic power of reversibility; in only a few cases is this ability lacking. By these simple facts much is explained that has been considered contradictory, unlikely, and even unexplainable in the literature on appendicitis.

A surgeon who is able to operate upon his cases of appendicitis within 48 hours can point to a low mortality—about 3 per cent. He will be an advocate of small incisions, preferably of "buttonhole" size. He does not recognize the need of getting rid of pus and of drainage ("the exudate is the body's reaction and as such is of positive benefit"), nor has he any difficulty with ileus, and he regards purulent appendiceal peritonitis as a simple disease in which diagnosis is often as simple as the operation and after-treatment. Those of us who have shared in the fight of the first pioneers against appendiceal peritonitis, and who have ourselves treated and unfortunately still have to treat late cases, understand those who complain of their difficulties with free appendiceal peritonitis in cases operated upon after 48 hours. The later cases come to operation, the worse the results.

One dealing with such cases must be content with a mortality of 20 to 60 per cent, must make use of long incisions, and must drain residual abscesses widely. To him it is obvious that, as in every case where an infectious and toxic pus is present, the pus must be removed and the cavity drained; to him the after-treatment with its symptoms of peritonitis ileus becomes a real crux. In every other case of ileus surgery is resorted to, often with brilliant results, but here one employs the whole arsenal of surgery and fails.

If we compare statistics based on the same principles and on similar material, we shall in all probability find that they agree. Should they be found to differ greatly, it will be due to circumstances of which we must be able to find the cause. It may be of interest to compare Bohmansson's statistics and my own (Table I).

TABLE I

Gangrenous Appendicitis without Purulent Peritonitis

	Cases	Deaths	Mortality Per Cent
Bohmansson (1935)	1,561	7	0.4
Giertz (1935)	2,000	19	0.8
Appendicitis with abscess			
Bohmansson (1935)	300	19	6.3
Giertz (1935)	568	37	6.5
Appendicitis with free (general) purulent peritonitis			
Bohmansson	472	68	14.4
Giertz, total number of cases	1,170	108	9.2
Operated on within 48 hours	839	29	3.5
Operated on after 48 hours	331	79	23.0

In passing I must criticize an expression used by many authors when dealing with the appendiceal peritonitis: namely, "perforation peritonitis." If they mean that the appendix is perforated in all cases with general purulent peritonitis, it must be pointed out that their experiences differ greatly from my own. Only about half of these cases present a perforated appendix.

It must be admitted that the two studies agree in a striking manner and differ no more than can be explained by physiologic variations.

On comparing my own material with that from the Uppsala Clinic of 1888 to 1907,[3] the result is quite different (Table II).

TABLE II

Purulent Peritonitis within 48 Hours

	Cases	Deaths	Mortality Per Cent
Uppsala material (1888–1907)	108	24	22.22
Giertz (1910–1934)	839	29	3.5
General peritonitis after 48 hours			
Uppsala material	100	53	53
Giertz	331	79	23
Localized peritonitis (abscess)			
Uppsala material	325	32	10
Giertz	568	37	6.5

It is at once obvious in Table II, how, in the course of time, operations have come to be performed at an earlier stage of the disease. Of the Uppsala material only 20 per cent of the cases were operated upon within 48 hours; of my own cases 50 per cent. The same tendency is also noticeable in my own material (in Umeå 33 per cent; at Sabbatsberg Hospital, 66.6 per cent).

It was from a study of the literature and the early Uppsala material that I came to understand the errors of Lennander's therapeutic method. It made use of large and numerous incisions, irrigations, enormous tamponades and a highly interfering after-treatment. On my own account and responsibility I began to follow almost entirely opposite principles, including small incisions, no unnecessary interference in the abdomen, and primary closure of early cases (as a rule, all cases within 48 hours). I admit that at first I did fall into the temptation of taking early active measures to prevent ileus. From this, however, I have departed more and more in relying on an expectant after-treatment.

The very considerable reduction from a mortality of 22.22 per cent to a mortality of 3.5 per cent among cases of purulent peritonitis operated upon within 48 hours can be interpreted in only one way: aggressive operative technic and therapy is greatly inferior to simple extirpation through small incisions and with primary closure. Nor is it difficult to find the explanation: the aggressive method with large incisions and drainage interferes in a disturbing manner with the development of Nature's own protective measures.

As to the treatment of walled off peritonitis, there has been no essential change in the method adopted, nor do the results show any improvement that cannot be attributed to earlier operation. In the treatment of general peritonitis after 48 hours I have also followed the new principles by using small primary incisions and opening residual abscesses as the need arises during convalescence. When necessary, drainage has been instituted, usually for abscesses with thick fetid pus and gangrenous walls. The improved results which I have obtained may be explained in part by this technic, but on the whole they are probably due to an earlier and more favorable operative material.

We then arrive at the third cause of the lack of mutual understanding on the question of peritonitis. When speaking of the treatment of these cases, we do not distinguish between operations undertaken for the sake of prophylaxis and those performed for the treatment of an already existing peritonitis and its sequences. In the infancy of abdominal surgery, when, as a rule, one had to face advanced cases with a peritonitis that had almost reached its terminal stage, there was no room for prophylaxis; it was the septic form of peritonitis and ileus with which one had to cope. It was therefore quite natural that the surgeon dealing with earlier stages of the disease brought with him all his views and preconceived ideas from moribund cases and treated early peritonitis as he had treated the late form. We have at last learned to abort a peritonitis by getting rid of the source of infection, and in the treatment of peritonitis already established to relinquish all active measures at operation. Experience has taught us that these so called prophylactic operations must be as simple and noninterfering as possible. However, if we are to carry out purposeful operations through small incisions, our diagnoses must be exact. We must avoid unnecessary exploratory incisions by all the means at our disposal.

When we remember the great possibilities of the roentgenogram in rapidly revealing stones, foreign bodies, free gas in the peritoneal cavity and ileus, it is easy to realize that such an examination has gradually become an absolutely essential aid to our diagnosis in acute cases of difficult interpretation. We must demand, however, that the examination, like all other necessary clinical proceedures, be carried out without delay. At the Sabbatsberg Hospital we now have a roentgenologist available at all hours. It may happen, however, that despite all examinations and a careful history, we do not get beyond a diagnosis of diffuse peritonitis of unknown origin. I have learned the advantage in such situations of making a small exploratory incision over the appendix, because appendicitis, "la grande maladie de l'abdomen," is, after all, the principal cause of peritonitis. Under all circumstances less damage is caused by a small exploratory incision over the appendix, even if later it is necessary to make an incision in the midline, than by an unnecessary midline incision in the presence of an extensive appendiceal peritonitis.

If one is confronted by the problem of peritonitis complicated by symp-

toms of ileus, one has to consider very carefully what may possibly be gained by active treatment. We would seem by now to have firmly established the impossibility of efficiently liberating a peritoneal cavity from infectious material either by mopping up or by irrigation, and the futility of attempting to drain the whole cavity. It is generally agreed that the only active measure that can safely be carried out in a peritonitis is the drainage and walling off by tamponade of certain parts of the peritoneal cavity, a method we use with success in solitary peri-appendiceal abscesses and residual abscesses after diffuse peritonitis.

In regard to ileus, it occurs in two different forms. One of them is the more or less typical ileus occurring when a loop of the small intestine adheres to itself and kinks until the proximal loop dilates and finally becomes completely incapable of peristalsis. This type occurs not only in forms of fibrinous peritonitis, but also in the initial stage of purulent peritonitis, and often in the case of encapsulated abscesses where coils of small intestine share in the walling off of the abscess. In all such cases of mechanical ileus in which the peritonitis is recent and acute, we must admit the possibility of an explanation of ileus other than the acute flexure. After all, in most cases we deal with a portion of an inflamed and infected small intestine in which the wall is toxically and mechanically affected, often transformed in effect, into a stiff and immobile tube.

The second form of ileus occurs in the fibrinopurulent type of peritonitis spread over the region of the small intestine. This form was previously called inhibited or paralytic ileus. It must now, however, be regarded as settled that the intestine is by no means, as was formerly believed, paralytic in its entirety. The loss of function is probably due partly to mechanical bends and partly to inactivity of certain portions of the jejunum and ileum from toxic or mechanical causes. There is still a great deal that needs explanation in the clinical picture of ileus. Thus, experimental work has been presented lately which tends to disprove the concept of specific toxins arising in the intestine from protein disintegration of the stagnated intestinal contents. Clinically, however, observations have been made that can with difficulty be explained without the assumption of such toxins. It has been definitely established that patients with ileus upon whom a Witzel gastrostomy is performed for the decompression of the upper parts of the jejunum and ileum experience immediate relief from symptoms of threatening circulatory collapse—a livid clammy skin and a faint, rapid, hardly perceptible pulse—symptoms characteristic of acute bacterial intoxication which are generally considered to be due to a peripheral affection of the capillary walls. If such a gastrostomy is closed before the distal obstruction has disappeared a rapid return of the symptoms ensues, with an equally rapid relief after the fistula is again opened. It seems to me that these indisputable facts are hard to explain without assuming the presence of a toxic substance, with a peripheral capillary effect, occurring in the upper ileum in the presence of stasis.

It is tempting for the surgeon to adopt operative measures against ileus.

In many cases of ileus with severe pain, fetal vomiting and a bad general condition, the life-saving operation which at once rapidly alters the clinical picture appears as a miracle. It is human that the surgeon, blinded by his success, should acquire a false belief in surgery as sovereign in all conditions of ileus. One soon learns that the treatment of ileus in cases with and without peritonitis are two fundamentally different things, partly because of the original disease, partly because of the difference between dealing surgically with a small intestine that is sound and one complicated by peritonitis.

During my period of training at Uppsala it was usual to employ a very active treatment for ileus, including gastrostomy, enterostomy, and even operative emptying of the small intestine. Cecostomy and ileostomy were frequently used, gastostomy later and to a less extent. In 1908 I analyzed in detail the material from the Uppsala Clinic and consequently warned against ileostomy. Even at the beginning of my independent work, therefore, I was skeptical about ileostomy, but in practice since then I have not wished to leave any means untried to give the patient a last chance and therefore have occasionally employed ileal fistulae, but only as a last resort. My results will be clear from the accompanying table, which includes a summary of all cases complicated by ileus in which an enterostomy was performed, of laparotomies accompanied by operative emptying of the small intestine and, finally, of all more major operations (Table III).

Altogether, 231 operations were performed. The annual average of operations of this type was at Umeå ten, at Stocksund ten, and at Sabbatsberg six. The average number of abdominal operations annually was: Umeå, 473; Stocksund, 777; Sabbatsberg, 765. Thus, despite an increasing number of cases, there has been a reduced number of operations for fistula, undoubtedly due to the fact that the clinical material has altered and the operative indications have become narrower.

I have divided my material from two main points of view: primary operative treatment for ileus, even at the first operation for the original disease, and secondary operations. Altogether 47 primary operations were carried out, of which there were no less than 34 at the Umeå Hospital, eight at Stocksund and only five at the Sabbatsberg Hospital. These figures indicate chiefly my altered views on the necessity and justification of the primary operation.

The second main group is concerned with operations for ileus in appendiceal peritonitis and ileus in other diseases. The first group is by far the larger, with 150 cases. The latter group includes 39 cases of peritonitis, the etiologic factor in which was other than appendicitis, and 42 cases of ileus without peritonitis. I have been unable to convince myself of the necessity of primary operations and, moreover, I have learned to understand how impossible it is to judge from the appearance of the peritonitis at the first operation whether or not it will be complicated by ileus. Without this qualification we operate on chance and carry out procedures which are often unnecessary and which may be harmful to the patient. This is particularly true as regards the emptying of the small intestine as suggested by Dahlgren.[4]

The primary major operations included in Table III concern all those patients with symptoms of ileus and walled off peritonitis in whom abscesses have been drained and intestinal obstruction excluded by means of anastomoses.

TABLE III

GASTRIC AND INTESTINAL FISTULAE, DRAINAGE OF THE SMALL INTESTINE AND OTHER OPERATIONS FOR RELIEF OF THE GUT

	Primary In Appendicitis	Fistulae In Other Diseases	Postoper. In Appendicitis	Fistulae In Other Diseases	Total
Umeå 1910–1921	19	15	48	39	121
Stocksund 1922–1927	8	..	33	22	63
Sabbatsberg 1928–1934	4	1	38	4	47
Totals	31	16	119	65	231

	Cases	Dead	Cases	Dead	Cases	Dead	Cases	Dead	Cases	Dead
Cecostomy	14	4	..	..	13	6	6	4	33	14
Gastrostomy	2	1	..	..	27	15	5	5	34	21
Gastrostomy and cecostomy	5	0	11	9	35	31	18	16	69	56
Operative emptying of small intenstine plus gastrostomy and possibly cecostomy	4	4	4	1	15	8	11	8	34	21
Operative emptying of small intestine and possibly cecostomy	2	0	..	..	3	2	12	10	17	12
Operative emptying of small intestine and ileostomy	..	..	..	..	1	0	..	..	1	0
Ileostomy	..	..	1	1	10	9	12	11	23	21
Second laparotomy with freeing of adhesions	..	..	..	..	4	0	..	..	4	0
Second laparotomy with ileo-ileostomy plus gastrostomy	1	0	..	..	2	1	..	..	3	1
Second laparotomy with ileo-transversostomy plus gastrostomy	3	1	..	..	6	4	1	1	10	6
Second laparotomy with resection of small intestine and gastrostomy	..	..	..	..	2	2	..	..	2	2
Second laparotomy with ileocecal resection and gastrostomy	..	..	..	..	1	0	..	..	1	0
Totals	31	10	16	11	119	78	65	55	231	154

As to secondary operations, ileostomy was performed in 23 cases in all, with 20 deaths. The patients saved by ileostomy were in two instances of pelvic abscesses following appendicitis, and in one instance fibrinous peritonitis without any demonstrable cause complicated by ileus. In the latter case cecostomy and gastrostomy were first performed, as well as emptying of the small intestine. Immediately ileus again supervened, at the second laparotomy the intestines were found adherent, expanded and friable. An ileostomy seemed the only feasible procedure, which subsequently caused marked maceration of the skin. Finally a third operation, resection of the intestine, resulted in ultimate recovery. In all other cases in which ileostomy had been performed, those with and those without peritonitis, the procedure proved quite useless. All the patients died despite the fistulae.

At last year's German surgical congress, Nordman[5] stated that he con-

sidered cecostomy to be superfluous. I am in agreement with him and have practically discarded this procedure. I have also abandoned the heroic measure of intestinal emptying which, particularly in peritonitis, must be considered a distinctly dangerous undertaking, not only because the intestine itself is usually friable and likely to suffer irreversible damage, but also because one may encounter unexpectedly an intra-abdominal abscess which may spread and cause an exacerbation of the peritonitis.

A Witzel's gastrostomy, on the other hand, is easy to perform. In contrast to the nasal catheter, with which I have had only slight experience, it causes little discomfort and generally closes immediately after removal of the tube. I think it is important that this tube, intended for drainage of the intestinal contents regurgitating into the jejunum, should be laid through the pylorus for some distance down into the duodenum. It happens that a gastrostomy thus made may very occasionally cause trouble, because the tip and side openings of the tube get caught by the valvulae conniventes of the duodenum. There have been patients in whom, despite a properly made fistula, nothing has passed either upwards through the fistula or downwards through the rectum, although at necropsy the small intestine was found dilated.

A definite effect of gastrostomy is the patient's objective as well as subjective improvement. He no longer suffers from fecal vomiting. With the fistula open he is able to take fluids without trouble, which is a very great relief. The feeling of oppression and, most important of all, the grave circulatory disturbances, disappear.

The number of cases is quite small for the evaluation of results—111 in all with a mortality of 72 per cent.

In appendiceal peritonitis.....	77 cases	54 deaths	71.4% mortality
In other forms of peritonitis and ileus.....	34 cases	24 deaths	72 % mortality

I am unable to say, as in the cases of ileostomy, that the gastrostomies were made on vital indications. Among those who did not succumb there were undoubtedly a good many who would have recovered without gastrostomy.

Among other major operations for ileus there were four cases of ileus from adhesions without a fatality. In all these cases there were severe inflammatory changes in the ileocecal region, especially of the lowermost part of the ileum, with adhesions which one neither could nor would wish to free.

The two cases of intestinal resection, both fatal, were hopeless. At operation for the obstruction such damage was caused to the intestines that only resection was likely to bring any relief.

In the last analysis it must be admitted that our ability to influence the progress of ileus by this method is very limited. *The only way of eliminating the difficulties is to improve prophylaxis* and this means *early operation for all abdominal cases that may be complicated by peritonitis.*

Table IV gives a brief review of the clinical material during this period (1910–1934). The number of abdominal operations was 16,815.

TABLE IV

Total Number of Abdominal Operations

Umea	1910–1921	6,825 cases
Stocksund	1922–1927	4,634 cases
Sabbatsberg	1928–1934	5,356 cases
Total		16,815 cases

Gangrenous Acute Appendicitis

Without purulent peritonitis	2,000 cases	19 deaths	0.95% mortality
With purulent peritonitis	1,728 cases	145 deaths	8.35% mortality
Totals	3,728 cases	164 deaths	4.4% mortality

Other Cases of Acute Peritonitis

Perforated gastric ulcer	149 cases	33 deaths	22.1% mortality
Gallbladder peritonitis	43 cases	14 deaths	32% mortality
Acute pancreatitis	33 cases	23 deaths	70% mortality
Acute general purulent peritonitis. Cause?	29 cases	15 deaths	52% mortality
Empyema peritonei and abdominal abscesses of unknown etiology	15 cases	.. deaths	0% mortality
Acute traumatic and other perforations of hollow organs	24 cases	16 deaths	67% mortality
Various	6 cases	2 deaths	
Totals	299 cases	103 deaths	34.4% mortality

There have been observed 2,027 cases of purulent peritonitis with 248 deaths (12.2 per cent). Peritonitis in connection with the female genitalia has not been included in this investigation. The majority of these cases are made up of purulent appendiceal peritonitis—1,728 cases with 145 deaths (8.35 per cent). All other forms of peritonitis amount to only 299 cases with 103 deaths (34.4 per cent). This latter group consists principally of perforated ulcers, 149 cases with 22.1 per cent mortality. There were in addition 43 cases of gallbladder peritonitis with a mortality of 32 per cent, 33 cases of acute pancreatitis with a mortality of 70 per cent, and 29 cases of ideopathic general purulent peritonitis with a mortality of 52 per cent. Fifteen cases were grouped under empyema peritonei and other abdominal abscesses of unknown etiology, with no deaths. There were 24 cases of acute traumatic and other perforations of hollow organs with a mortality of 67 per cent, and of more unusual forms there were six cases and two deaths. Four of these six cases proved to be actinomycosis with only one death.

The overshadowing importance of appendicitis in the abdominal material that is encountered by the Surgeon-in-Chief of a community hospital in Sweden becomes still more obvious if one remembers that beyond these 1,728 cases of purulent appendiceal peritonitis there have been under treatment

2,000 cases of gangrenous appendicitis with or without only serous fibrinous peritonitis, with 19 deaths (0.95 per cent mortality). Finally, there are still 1,633 cases of acute non-destructive appendicitis with eight deaths (0.50 per cent). The sum total of appendiceal cases thus amounts to 5,361 with 172 deaths (3.2 per cent) (Table V).

TABLE V

ACUTE APPENDICITIS

	Umeå 1910–1921		Stocksund 1922–1927		Sabbatsberg 1928–1934		Total			
	Cases	Deaths	Cases	Deaths	Cases	Deaths	Cases	Deaths		
I. Without purulent peritonitis										
A. Nongangrenous acute appendicitis	800	2	328	1	505	5	1,633	8	0.5%	mortality
B. Gangrenous appendicitis	493	6	439	5	1,068	8	2,000	19	0.95%	mortality
II. Gangrenous appendicitis with purulent peritonitis										
A. Early peritonitis < 48 h.	224	5	216	8	399	16	839	29	3.5%	mortality
B. Late peritonitis > 48 h.										
a. free general	125	36	101	25	95	18	321	79	23%	mortality
b. walled off	334	20	128	11	106	6	568	37	6.5%	mortality
Totals	683	61	445	44	600	40	1,728	145	8.35%	mortality
Sum totals	1,976	69	1,212	50	2,173	53	5,361	172	3.2%	mortality

In the foregoing I have dealt with *appendiceal peritonitis.* When we urge early operation we imply an early and exact diagnosis; without such we come to slaughter "en masse" or to extirpation of appendices for reasons other than the patient's health and safety.

Two further methods of investigation of great assistance in diagnosis have come in recent years to be used in every acute abdominal case: the white blood count and determination of the sedimentation rate. These examinations have been carried out routinely at the Sabbatsberg Hospital since 1928. In the cases of acute appendicitis the material has been studied by one of my assistants. Although I am unable as yet to submit any exact figures, in general the results of this investigation are that in acute gangrenous appendicitis an increase in the number of leukocytes, generally to 10,000–14,000 per cm. and not infrequently above 20,000, takes place in by far the greatest number of cases. The sedimentation rate, on the other hand, is normal during the first few days. If, therefore, in a patient with acute abdominal pain we find a high sedimentation rate and a low leukocyte count, the appendix is probably not the source of the peritonitis. And the contrary holds true: an acute abdominal case with a typical history of appendicitis, even without a rise in temperature, increased pulse rate and abdominal tenderness, but with a low sedimentation rate and a high leukocyte count, is operated upon for destructive appendicitis.

However experienced we become in the course of years and however con-

scientious we are in our observations, we have to face almost daily patients with acute abdominal pain on whom we are unable to make an exact diagnosis and in whom we are unable to exclude the possibility of appendicitis. In such cases I perform, on principle, an exploratory laparotomy over the appendiceal region.

For a long time I have been trying, clinically and statistically, to find an answer to several important questions which must be solved:

(1) How often do we make a diagnosis of acute gangrenous appendicitis and yet find a normal appendix at operation?

(2) How often do we operate with an uncertain diagnosis, believing acute appendicitis cannot be excluded, and find a gangrenous appendix at operation?

(3) How are we to explain the symptoms if, at such exploratory laparotomy, the processus verimformis is found to be perfectly normal? As we all know, a great number of patients exist, particularly women, who subjectively present such suspicious symptoms of appendicitis as to demand an exploratory laparotomy, but in whom operation reveals nothing.

(4) What risks do these patients run if submitted to an exploratory laparotomy and how great are these risks as compared with the risk of nonoperative treatment?

That all risk is not absent in the removal of an appendix, even when there are no signs of gangrene, is also evident from the eight deaths among 1,633 operated cases, a mortality of 0.5 per cent (Table VI).

In five cases the cause of death was a pulmonary embolus which occurred no less than 17 times in the whole series, and in one case the cause of death was a technical error. Extirpation of the appendix in the free interval or while in a mildly inflammatory stage without peritonitis is one of the easiest abdominal operations, and can safely be carried out by young and relatively inexperienced assistants. However, when the symptoms are more than 48 hours old one can never be certain what surprises lie in wait for one. For a long time, therefore, I have allowed no one but the two senior assistants or myself to operate upon patients with peritonitis and acute appendicitis who have been ill for more than 48 hours.

The two other deaths occurring in acute nongangrenous appendicitis were caused by septicemia from a widespread phlegmon of the abdominal wall. In both cases the appendix was unfortunately not examined microscopically. For many years we have verified all appendices microscopically. Experience has taught me that appendices regarded as grossly as "gangrenous in the mucous membrane" have often presented microscopically no such changes, and appendices that appeared innocuous at operation have not infrequently been found greatly altered on microscopic examination.

If we adopt primary closure of the wound as an arbitrary principle in the treatment of peritonitis, experience must soon tell us whether or not there is any inherent danger in the procedure. This danger presented itself in my series as a severe phlegmon of the abdominal wall, which required

secondary incisions (31 cases). In eight cases in which no peritonitis was found this plegmon of the abdominal wall so dominated the whole clinical picture that death must be regarded as the result thereof. In the other fatal cases (five) there was at the same time a widespread purulent peritonitis, making it difficult to assess to what extent the abdominal phlegmon was the cause of the fatal issue.

TABLE VI

CAUSES OF DEATH IN APPENDICITIS

	Without Purulent Peritonitis		With Purulent Peritonitis			Totals
	Nongangrenous	Gangrenous	<48 h.	>48 h. Free	>48 h. Walled off	
Complications						
Pulmonary emboli	5	5	2	1	4	17
Technical operative error	1	..	..	..	..	1
Pneumonia	..	1	..	2	2	5
Lung abscess	..	1	..	..	1	2
Synechia pericardii	..	..	..	..	1	1
Tbc. pulm. miliaris	..	..	1	..	1	2
Cerebral hemorrhage	..	..	..	1	..	1
Totals	6	7	3	4	9	29
Peritoneal Infections						
Septicemia	2	6	..	..	8	16
Peritonitis	..	..	4	32	4	40
Peritonitis with ileus symptoms	..	..	17	30	5	52
Paralytic ileus	..	..	4	3	..	7
Mechanical ileus	..	4	..	1	8	13
Healing peritonitis with ileus symptoms plus pneumonia	..	..	..	6	2	8
Healing peritonitis with ileus plus thrombosis of vena cava	..	..	..	1	..	1
Purulent pericarditis	..	..	..	..	1	1
Mesenteric venous thrombosis and liver abscess	..	2	1	2	..	5
Totals	2	12	26	75	28	143
Sum totals	8	19	29	79	37	172

Residual abscesses must also be considered. The commonest of these is the pouch of Douglas abscess which occurred in 123 cases with 11 deaths. If we realize that most of these abscess operations were carried out in cases of appendiceal peritonitis of widespread character, it is clear that the production of a pelvic abscess indicates a good reaction of the body and is, therefore, a favorable sign. There are other intra-abdominal residual abscesses

that often must be opened. Nonsubphrenic abscesses, usually located in the left iliac fossa, have been encountered in 38 cases with four deaths. Subphrenic abscesses occurred in 11 cases with three deaths.

For the sake of completeness I would like to report on subphrenic abscesses arising from causes other than appendicitis (Table VII).

TABLE VII

SUBPHRENIC ABSCESSES RESULTING FROM OTHER THAN APPENDICITIS

In infection of the gallbladder	3 cases	1 death	1 case not operated upon
In gastric ulcer and perforated duodenal ulcer	5 cases	4 deaths	
In gunshot wound of the stomach	1 case	0 deaths	
In rupture of the liver	1 case	1 death	
In retroperitoneal tumor	1 case	1 death	
After operation for fibrous peritonitis	1 case	1 death	
Cause unknown	1 case	0 deaths	
	13 cases	8 deaths	

The tabulation of the causes of death in purulent peritonitis includes only well known conditions. In this connection, it may be mentioned that the total number of observed cases of liver abscess in cases of appendicitis amounted to six with as many deaths. In two patients, both dead from concurrent peritonitis, the abscess was discovered at postmortem. Incision and drainage were carried out in the other patients. There was only one liver abscess with an origin other than appendicitis and this followed perforation of a gastric ulcer.

In regard to perforated peptic ulcer, there was an annual average of three to four cases at Umeå, six at Stocksund, and ten in Stockholm (Table VIII).

The improved results in recent years may be due in part to a more correct, more certain and more consistent selection of operative methods, but to a large extent it is explained by the fact that in recent years in Stockholm patients have been reaching us in an operable condition. They are now sent directly to the hospital at the onset of pain. The primary principle to be borne in mind is that the peritonitis must be treated before the ulcer disease. Should the patient not be in good condition or if more than four hours have elapsed since the perforation, if there should be any contra-indication to a major operation after exploration of the abdomen or if the operator is insufficiently experienced to perform gastro-enterostomy or resection with ordinary risk, nothing is done save closure of the ulcer, possibly after excision of its margins and a Witzel gastrostomy. In these cases in which the gastrostomy is intended only for relief of the stomach, the tube is placed with the tip toward the cardia and the abdomen is primarily closed. In the course of the last three years, 1932 to 1934, we have operated upon 27 cases with only one death. The single fatality was a patient who had been ill for seven days and was beyond surgical help. Only incision and drainage were carried out. In the other 26 cases gastro-enterostomy was performed on two

patients and resections on two; in the remainder only suture and gastrostomies were performed.

TABLE VIII

PERFORATED GASTRIC AND DUODENAL ULCER

Operative Method	Umeå 1910–1921	Stocksund 1922–1927	Sabbatsberg 1928–1934	Totals	Mortality
Suture of ulcer with primary closure of wound (usually gastrostomy)	10–4	9–5	49–9	68–18	26.5%
Suture of ulcer and gastro-enterostomy with primary closure of wound (usually gastrostomy)	28–7	24–4	9–0	61–11	17%
Resection with primary closure of wound (usually gastrostomy)	1–0	4–1	9–1	14–2	15%
Drainage without suture of ulcer with gastrostomy	3–0	....	1–1	4–1	
Operation for perforated jejunal ulcer	1–0	....	....	1–0	
Drainage of perigastric abscess	1–1	....	....	1–1	
Totals	44 12	37 10	68 11	149 33	

CAUSES OF DEATH

	Umeå 1910–1921	Stocksund 1922–1927	Sabbatsberg 1928–1934	Totals
Peritonitis—septicemia ileus	7	7	9	23
Postoperative shock, 1; pneumonia, 5; lung gangrene, 1; circulus vitios following gastro-enterostomy, 2; volvulus, 1	5	3	2	10
Totals	12	10	11	33

The cause of death in 23 of the 33 fatalities was a direct result of peritonitis (septicemic peritonitis—ileus). In ten cases (30 per cent) death was due to causes not directly connected with the disease as such. In no less than nine of these ten cases death must have been related to the operation (Table VIII). During my tenure as assistant to Lennander a perforated ulcer that could be saved was almost a miracle; in a report on cases published in 1898 only two out of 11 cases survived, a mortality of 82 per cent. If we take into consideration the large incisions used at that time and all attempts at cleaning and drainage of the abdominal cavity, as well as interfering after-treatment, and compare this with our present simple operative method and expectant after-treatment, it must surely be admitted that figures and analyses of cases speak a language that cannot be misunderstood.

In the course of the past 25 years 1,017 gallbladder operations have been

carried out with 96 deaths, a mortality of 9 per cent. Only 43 of these cases were complicated by purulent peritonitis; in 26 the peritonitis was general, in 17 walled off, and 14 died (33 per cent). My figures show how the gallbladder material in these years has increased in our hospitals. During the Umeå period there was an annual average of ten cases, at Stocksund of 55, and at Sabbatsberg of 82. I have not prepared this material sufficiently to be able to give a detailed account of the significance of the figures. However, it is clear that a diseased gallbladder, even if highly gangrenous, rarely gives rise to a peritonitis, and that this peritonitis is as a rule mild in character. Another characteristic feature of gallbladder peritonitis is its rare complication with ileus. The high death rate is not usually due to the peritonitis, as such, but to the original disease, inflammation of the bile tracts and calculi, which, in those cases that lead to peritonitis or pericholecystic abscess, is complicated and difficult to treat.

A disease about which a great deal is written, although I have had difficulty in reaching a real understanding of it through studies of the literature, is *pneumococcal peritonitis*. Nor has my experience with this disease brought me any enlightenment. In the course of 25 years I have seen three cases of walled off umbilical abscesses (so called empyema peritonei). The clinical features of these cases have agreed with those described as characteristic of a chronic form of pneumococcal peritonitis. These abscesses have been of benign nature and have cleared up after incision.

During the same period I have further observed 29 cases of acute purulent peritonitis in which neither at operation nor necropsy (15 deaths) could any local cause of peritonitis be found. Unfortunately, in none of these cases was a very careful bacteriologic examination possible. In two cases, both dead, I have ventured the diagnosis "probably pneumococcal peritonitis." In three cases the peritonitis was labeled metastatic and in two cases it probably arose from a simultaneous tonsillitis; in the latter the course was very acute. All cases were operated upon, since I believe that a gangrenous appendicitis can never be ruled out without operation.

In the past 25 years three cases of gangrenous appendicitis have been admitted in good time for a prophylactic operation, but because of mistaken diagnoses operation was not carried out until too late, after aggravation of the symptoms had made operation necessary. Diagnoses of pneumonia were made in two cases and salpingitis in the other.

In all the 29 cases just mentioned the appendix was macroscopically as well as microscopically normal. In a fatal case of peritonitis in a girl the tubes and uterus were found to be microscopically normal. In eight patients, five of whom recovered, a normal appendix was left to avoid unnecessarily prolonging the operation. Whether this procedure darkened the prognosis of any of those 15 patients who died after the operation I do not know, but there is no evidence to support such a suggestion. I am quite convinced that these 29 cases may be referred to widely different types of peritonitis.

Thus in our work we occasionally meet with cases of acute purulent

general peritonitis in which we are unable definitely to establish the etiologic factor. Some of them are undoubtedly of the pneumococcal type, and others metastatic (in connection with purulent pleurisy and pericarditis). I believe it is evident from my statistics that there is little harm in exploratory laparotomy in these cases.

There is a disease of childhood, however, *during which exploratory laparotomy in the acute stage is undoubtedly an operation to be avoided: namely, scarlet fever.* Two female infants who had been taken acutely ill with fever, vomiting, and diffuse abdominal pains, and who had been admitted to the hospital without any thought of scarlet fever, were subjected to exploratory laparotomy on a presumptive diagnosis of acute gangrenous appendicitis. In both cases the appendix proved blameless and there were no signs of peritonitis. In both a severe form of streptococcal peritonitis developed which led to death in one. The other girl recovered but only after prolonged suffering and a number of incisions for residual abscesses in the abdomen and below the diaphragm.

All the localized abscesses in the abdomen which were without ascertainable starting point[12] proved to be benign. In one case there were multiocular abscesses with ileus in which 190 cm. of the small intestine had to be resected. Recovery followed. In two cases of abscesses in the right iliac fossa and the right lumbar region quite normal appendices were removed. These lay outside the abscesses concerned (Table IX).

TABLE IX

Perforated Injuries from External Violence

Gunshot wound of stomach	2 cases	0 deaths
Gunshot wound of colon	3 cases	1 death

Subcutaneous Perforations

Rupture of biliary tract	1 case	0 deaths
Rupture of ileum	4 cases	4 deaths
Rupture of colon	2 cases	2 deaths
Totals	12 cases	7 deaths

In perforation of diseased hollow organs the original disease was:

Typhus, 2 cases; tbc, 1; hernia, 2; infected tumor, 2	7 cases	5 deaths
Diverticula coli	4 cases	4 deaths
Meckel's diverticulum	1 case	0 deaths

CONCLUSIONS

We must realize that we cannot treat acute peritonitis according to a set pattern or by a fixed method for all cases. We must learn to treat peritonitis methodically; that is to say, we must realize that there is not one form of peritonitis but many, each with its special etiology, peculiar symptomatology and specific pathologic anatomy. Naturally, all have well marked features in common because they occur in the same human body.

We must realize that cases of purulent peritonitis demand the most accurate diagnosis, because on this the treatment depends, and in peritonitis more than in any other disease treatment is a life or death matter. While one case may demand a rapid and purposeful surgical intervention, another may require an equally purposeful nonsurgical measure. A mistake on the part of the surgeon may cost the patient his life. It must be understood that one type of peritonitis in different stages of its development presents various clinical pictures and therefore requires different forms of treatment.

First and last we must remember that, even with the aid of surgical and other means, our chances to help a patient with peritonitis in advanced stages are very small. The victories we have gained in the course of the past twenty-five years, and which we are able to estimate statistically, are due to our understanding of the prevention of peritonitis through early prophylactic operations. For further success we must proceed along the same course, which means continued enlightenment of the public regarding the very great risk of life inherent in delay in the treatment of acute abdominal pain.

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Discussion.—Dr. Franz Torek (Montclair, N. J.).—I think we are to be congratulated upon having heard Professor Giertz' exposition of this condition. As he has mentioned, the surgeon is often confronted with the fact that at the time when he is called in it is too late for prophylaxis, but the surgeon has to interfere immediately.

As for the method of interference, I think we are all agreed upon certain points, namely, that if the condition is either localized by adhesions, or if it is a free peritonitis of moderate extent, we confine our interference to the part that is involved, being careful not to spread it any further. We accomplish this by walling off the affected area and avoid touching those parts which are still uninvolved.

It is different if we have to deal with a diffuse suppurative peritonitis, especially that following appendicitis. By the term "diffuse" I mean a condition in which the suppurative peritonitis has extended well over to the opposite side, and high up. In reference to this class of case I wish to call attention to 36 case reports cited many years ago, which were seen early in my career. Later on, the diffuse cases became more and more rare, which of course I believe is due to the fact that we recognize the condition earlier and operate earlier.

I shall not go into all the details, but refer you to the transactions of this society for 1932, page 69. I should like, however, to consider some of the principles involved. It is important that all the pus must be evacuated. If the procedure is such as to give only partial relief from the infection, with

considerable accumulations of pus left behind, I think the patient is usually doomed. That such pockets may exist I learned very early in my observations. After allowing all the pus to flow out, I would find adjacent coils of intestine behind which large amounts of pus were concealed. All portions of the peritoneum must be reached, and the only way I felt I could accomplish this was through a large incision. From pubes to umbilicus is not sufficient. The incision must go higher than the umbilicus, affording access to all the intestine, so as to permit handling them as gently as possible. This can best be effected by a stream of water. I have been employing large quantities of saline solution, aiding the lavage with the gloved hand to move the intestines very gently from side to side, to be sure it will reach all parts.

That, of course, is done in a certain order, after the opening is made and the free pus evacuated, sometimes in enormous quantities. I recall one case in which, at the first nick in the peritoneum, the pus spouted up a foot or more. By the way, that was one of the successful cases.

The orderly procedure would be first to go into the region of the appendix, to remove it as quickly as possible and wash out that gutter. Next wash out the pelvis and after that clean the left gutter. After everything is grossly clean, and the water returns clear, I believe the peritoneum will be capable of coping with any residual infection.

After this is accomplished, the abdomen is closed without drainage. That is important, I believe, because whatever drainage might result can only do harm. You cannot drain the entire peritoneal cavity. Consequently, you have to depend upon the ability of the peritoneum to overcome what infection is left.

As to my results: I published them in two sections, each one detailing 18 cases, and the results in both groups were the same; namely, one death in every six patients, making a mortality of 16.66 per cent. Subsequent to the compilation of the above statistics I operated upon four other cases without a death, which reduces the mortality to 15 per cent.

Dr. Alton Ochsner (New Orleans, La.).—Whereas the mortality rate immediately after the production of a peritonitis may be due to the absorption of toxin by the peritoneum, those cases that live after the peritoneal reaction has occurred die mostly of the ileus which Professor Giertz referred to. The adynamic ileus can be readily and easily combated by relatively simple measures, such as the decompression suggested by Wangensteen with the indwelling duodenal catheter and suction, and the administration of large doses of morphine as suggested by Orr and Clark. Doctor Orr has presented before this Association his results, showing that morphine is a stimulant to intestinal activity rather than an inhibitant. If one will give these patients large doses of morphine and keep them well narcotized until their peritoneal reaction has subsided, their ileus will largely be taken care of.

Several years ago we began using oxygen, with the idea of combating an anoxemia, and our results are gratifying. Recently Fine, of Boston, has shown that the inhalations of oxygen are beneficial in hastening the absorption of retained gas in intestinal loops. Personally, I feel that this is a great contribution, and I feel that these patients with intestinal ileus, of any type, irrespective of the cause (and in these cases it is usually the adynamic variety) should be given large doses of morphine and oxygen.

In addition to this, the application of heat to the abdomen is of value, probably because, as Mueller has shown, it produces a vasodilatation of the somatic vessels and a constriction of the splanchnic vessels, which in turn favors the relief of the ileus.

Dr. Owen H. Wangensteen (Minneapolis, Minn.).—Certainly Professor Giertz should be commended for this discerning and well tempered dissertation relating to peritonitis; and I find myself in practical accord with the principles which he has enunciated. He has alluded to the fact that in the presence of peritonitis, the surgeon not uncommonly does harm and spreads the peritonitis by badly timed operative intervention. This is an experience which I have also had. We have lived through a few decades, during which time "acute abdomen" has been synonymous with operation. We now stand, I am inclined to believe, on the brink of a new era in abdominal surgery, as it relates to the acute abdomen, in which one will not arbitrarily operate because an acute abdominal lesion is present. The criteria which *demand* operation must be defined; no longer is "acute abdomen" to be considered synonymous with necessity for operative intervention. Operation is to be undertaken only on adequate indication and when it is believed that something may be accomplished by it.

I would agree that every patient with acute appendicitis should be operated upon early. In instances of perforation of the gastro-intestinal canal, whether of the stomach, small intestine, or colon, operation is urgently indicated to close the leak. In all strangulating types of obstruction and in acute obstructions of the colon with considerable distension, immediate operation is in order. Experience has adequately shown, however, that frequently in mechanical obstruction of the small bowel due to adhesion, decompression can be effected through the agency of suction applied to an inlying duodenal tube without recourse to operation. I see very little necessity for frequent operative attack upon the so called acute gallbladder. Practice has shown, in the main, that conservative, nonoperative treatment of biliary colic is safe. To be sure, an occasional case will demand operation. Those who frequently practise and urge excision of the "acute gallbladder" are apparently unmindful of the fact that the gallbladder, when obstructed, is not as treacherous as is the appendix. It is my belief that if one took two parallel series of 100 "acute gallbladders" and operated upon all in one, and none in the other, that there would be more lives saved in the group in which operative restraint was practised. If in addition, in the conservatively treated group, one individualized the cases after careful scrutiny and urged operation upon those where it was manifestly needed, the salvage of life would be further increased.

Moreover, I see but slight indication for operation in instances of pancreatic necrosis. The diagnosis in most instances can be made without operation with some assurance on the criteria which I have elsewhere described. (Minn. Med., **15**, 201, March, 1932.) The operative measures which have been employed in this condition are essentially three: (1) drainage of the peritoneal cavity, (2) drainage of the biliary tract, (3) tamponade of the pancreas. None of these is in any sense a specific remedial measure. Patients die of pancreatitis because of autodigestion of the pancreas. What is needed is some agency which will interfere with the activation of trypsinogen within the pancreas. Partial removal of the normal activator of the pancreatic ferments, succus entericus, from the lumen of the duodenum by suction, starvation, and the administration of atropin are probably just as effectual in this respect as are the nonspecific operative procedures commonly employed.

Professor Giertz has rightfully stressed the importance of an adequate evaluation of the findings of the physical examination of the abdomen in acute abdominal lesions. Despite the fact that "abdomen" means hidden, it is astounding how accurate diagnoses can be when proper attention and

significance are accorded the history, inspection of the abdomen, palpation, percussion, auscultation, and the roentgenologic findings in acute abdominal disorders. With improved diagnosis, and mindful of the conditions which can be remedied by operative intervention, as well as of the shortcomings of the surgeon's art, a more conservative policy in dealing with acute abdominal afflictions will probably become generally apparent. The frequent employment of suction to prevent and combat distension which so commonly attends acute introperitoneal lesions, widens the scope of conservative management. We must critically examine ourselves and our therapeutic remedial measures lest we arrogate to ourselves, or attribute to them, credit which neither we nor they deserve.

Dr. Vernon C. David (Chicago, Ill.).—I greatly enjoyed Professor Giertz's remarks, and his statements, of course, reflect a great deal of very practical experience in this field.

It seems to me, in a simple way of looking at it, that one of the most important things about operation for peritonitis is to close a continuing contamination from any source within the peritoneum. Once that is done, the main object is obtained. It can be shown very easily, experimentally, that the exudate, is really a protective mechanism, and that this exudate really prevents absorption of toxins as well as bacteria from the peritoneal cavity.

One of the important considerations about the mortality in peritonitis concerns itself with the occurrence of ileus. The development of ileus can be very little interfered with, or very little controlled by operative procedures, except those which close the source of contamination, because most of the cases of ileus result primarily from damage to the bowel wall itself by inflammation with resulting loss of peristalsis, and the main thing that can be done after such a mechanism has been established is to keep the intestinal tract as free as possible by continuous suction or other means embodying that principle.

It is a well known fact, for instance, that when a bowel is brought out onto the abdominal wall for ileostomy or colostomy, it takes only a few hours before the inflammation of that loop causes the loss of all its peristaltic activity, but when that inflammation is controlled, the bowel again takes on its functions, and I believe personally the same thing holds true in regard to the formation of ileus as in most cases of peritonitis.

Dr. Rudolph Matas (New Orleans, La.).—I cannot see this discussion come to a close without a note of personal appreciation of the distinguished speaker, Professor Giertz and his Swedish colleagues. Those of us who have had the good fortune to visit the hospitals and medical institutions of Stockholm will always hold a delightful recollection of the unfailing courtesy and generous hospitality of the master surgeons of that city, whom he so fittingly represents.

No one who has attended the surgical clinics of Stockholm, Upsala and Lund can fail to be impressed with the great ability, conscientious thoroughness and alertness for every advance, that characterize the Swedish School of Surgery and that make its contributions to surgical progress and literature so highly esteemed by the surgical world. It is, therefore, especially gratifying to welcome Professor Giertz, and to renew here, as on a previous occasion, the cordiality of our greetings to an honored guest.

In the paper we have just heard, Professor Giertz has given us the results of his ripe and varied experience in the prevention and treatment of peritonitis, a subject which in its many and varied etiologic aspects continues to furnish perennial material for profitable discussion. His conclusions fully

confirm what is generally agreed, that once a fully developed septic peritonitis is established, we are dealing with one of the greatest causes of surgical mortality. We are also generally agreed that the great advances made in reducing the mortality of peritonitis are due to prevention by earlier diagnosis and prompt surgical removal of the potential or actual causes of peritonitis, which ever they may be, rather than by any measures of treatment adopted after diffuse peritoneal sepsis has asserted itself. We need go no further than the story of appendicitis to confirm these generalizations. Doctor Torek's picture of appendicitis, as it appeared to us 40 years ago and as we see it now, especially appeals to me as a contemporary who has lived through the same experience. It may be truly said that for nearly two decades after Fitz had identified the appendix as the chief cause of peritonitis and its complications, we rarely, if ever, operated for appendicitis as a strictly intra-appendicular disease; it was always after perforation and gangrene had infected the peritoneum and caused either a localized suppuration or a generalized peritoneal invasion with all its disastrous consequences. Trust in medical treatment and the fear of postoperative complications invariably delayed the operation long after a preventive or timely appendectomy could be performed. The best that could be expected was a well walled abscess which could be drained with or without the extirpation of the appendix, often leaving protracted fecal fistulae and disabling ventral herniae. The diffuse suppurations, complicated by migratory absecesses, paralytic ileus with uncontrollable vomiting of duodenojejunal contents, were common and the yellow fever type of appendicitis, caused by portal thrombophlebitis, with typical black vomit and jaundice (now practically unknown), was not rare. Bold attempts at free peritoneal irrigation with multiple drains only hastened the fatal termination. No wonder that under these tragic circumstances, all aggressive surgery was abandoned and the old classic treatment of peritonitis by absolute gastro-intestinal immobilization with opium, starvation and rectal drips, popularized by J. D. Ochsner, came into vogue, leaving to surgery only the task of draining localized suppurations.

This method at least tended to arrest the spread of the infection and yielded better results and was less discomfiting to the surgeon and his reputation.

Now, all this has changed. Thanks to the intense, nationwide campaigns conducted against the appendix—day in and day out—by medical societies, popular lectures, the lay press, radio talks and by other innumerable agencies for the diffusion of medical information, the public at large has been thoroughly informed of the dangers of delayed operations and indiscriminate purgation, resulting in the fact that we no longer have to beg patients to have their appendices removed. Now, the mere mention of appendicitis suffices to hasten the alarmed patient to the hospital and the operating table. In this way the number of prophylactic appendectomies have greatly increased and the mortality has unquestionably diminished, despite the contradictory statistics that have appeared on the subject. The fact is that the surgeon today is far more comfortable in the presence of appendicitis and that the tragic specter of septic appendicular peritonitis is fast retreating into the shadows of a grim past, to be replaced by the smiling faces of countless convalescent aseptic appendectomized patients. As in all popular educational campaigns intended to warm the public against avoidable dangers, an alarm is created which is only too frequently abused. But while I am unconditionally opposed to any and all operations performed without a reasonable foundation, I would rather see a barrel full of normal appendices removed through honest error, than see a single avoidable death caused by neglect of

timely surgical intervention. While hailing the advent of a new era of prophylactic appendectomies, I am not blind to the fact that, unfortunately in too many instances, the first intimation of appendiceal mischief coincides with a perforation and peritoneal invasion. When this happens, even an immediate operation will disclose that peritoneal infection has occurred and that the door to septic, secondary abdominal complications has been opened. The extirpation of the appendix and exclusion of the primary focus undoubtedly improve the patient's chances, but when widespread infection has occurred, the old tragic picture of diffuse peritoneal sepsis is revived. Then a new chapter of anxious complications begins which, though gloomy, is not as hopeless as in the past, and still permits the display of lifesaving resources not available, or thought of, until relatively recent years and that often will tide the patient over a seemingly fatal crisis.

In referring to great improvements in the treatment of the postoperative complications, the speaker had in mind at least two contributions which have proved of the greatest value in dealing with adynamic or paralytic ileus—one of the most frequent and formidable manifestations of peritoneal infection.

First: the relief of the uncontrollable and exhausting regurgitant vomiting by the method of continuous drainage of the gastro-intestinal contents by syphonage or suction, through an indwelling duodenal tube introduced through the nasopharynx, and allowed to remain *in situ* as long as the regurgitation of the gastro-duodenal contents continues, thus permitting unrestricted drinking of water or other refreshing fluids *per os,* while the drainage is going on automatically. The stomach in the meantime undergoes frequent lavage, while gases and noxious gastro-intestinal secretions are being continuously and unconsciously evacuated. The duodenal tube here acts as an artificial anus, *per vias naturales,* and effectively prevents dilatation and autocompression of the stomach and upper gastro-intestinal loop.

Second: The continuous infusion of dextrose and salt solution by the intravenous drip (phleboclysis), which provides against dehydration, starvation, dechlorination and arterial hypotension while permitting medication with cardiovascular stimulants, and alternating transfusions of whole or citrated blood as may be indicated to reinforce a failing heart and circulation.

These two procedures which were first introduced and adopted by the speaker 23 years ago, have been fully described (Transactions Am. Surg. Assn., 41, 468–491, 1923; Annals of Surgery, 79, 643–661, May, 1924) and have since come into general use—more or less modified and under different names. These two methods alone, when combined with the free use of morphia, have often sufficed to tide the patients over a dangerous crisis, giving the natural defenses a chance to assert themselves in restricting the spread of the infection and in restoring the normal balance of gastro-intestinal function. Jejunostomy remains, none the less, a supplementary measure of most decisive and lifesaving importance when performed at the proper time and in the proper way, before the patient's vitality has been completely exhausted.

Summary.—(1) While the number of operations for appendicitis have greatly increased, the mortality of the operation has very notably diminished. The lowered mortality is due chiefly to the general acceptance, by the public and the profession, of the principle of early prophylactic operation in the intra-appendicular stage of the disease and before peritoneal contamination has occurred.

(2) The very great improvement in the treatment of the postoperative complications, when peritoneal contamination and infection has already occurred.

THE PREVENTION OF PERITONEAL ADHESIONS BY PAPAIN

A CLINICAL STUDY

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ALTHOUGH *fibrinous* peritoneal adhesions are in most instances protective and therefore desirable, especially in the presence of peritoneal infection, the development and persistence of *fibrous* adhesions are often distinct menaces to the comfort, health, and life of a patient. Whether intra-abdominal adhesions are the result of mechanical, thermic, chemical, or bacterial trauma to the peritoneum, they are generally directly proportionate to the degree, extent, and persistence of the peritoneal injury; therefore, the adhesions following bacterial trauma are usually more marked than those resulting from the less persistent traumatizing mechanisms. Whereas the fibrinous, useful serosal adhesions accompanying peritoneal infection usually disappear after the subsidence of the bacterial invasion, occasionally they persist and become transformed into fibrous adhesions. Infrequently, when damage to the peritoneum is not associated with infection, fibrinous adhesions which have served little or no purpose also become organized and may produce disturbances in intestinal motility. Normally, after the subsidence of the peritoneal infection the proteolytic ferment derived from leukocytes in the peritoneal exudate causes the fibrinous peritoneal adhesions to disappear by a process of autolysis. The reason for the failure in some cases of the normal disappearance of the fibrinous adhesions is not known, but probably in such individuals there exists an abnormal tendency toward the development of fibrous tissue, *i.e.*, "keloid tendency" or "adhesion diathesis."

As peritoneal adhesions are the result of trauma to the serosa, it is imperative that the surgeon minimize the trauma as much as possible by avoiding mechanical injury, cooling and dehydration of the peritoneum during the operation, chemical irritation, and the introduction of microörganisms from without or from the viscera. Whereas the necessity of observing these principles is obvious and, by so doing, the incidence of adhesions can be materially decreased, infection is sometimes already present and, furthermore, considerable peritoneal mechanical trauma may be an unavoidable accompaniment of the operative procedure. In such instances, relatively little can be done to prevent the formation of adhesions, which, as a matter of fact, are desirable and protective. In those individuals in whom the adhesions persist, become organized, and produce disturbances in intestinal motility, subsequent operations become necessary in order to divide the adhesions. Meticulous care during the operative division of the bands both as regards the minimizing of mechanical trauma and the prevention of cooling and infection can

never be supplanted by other measures to decrease the incidence of adhesions. There are, however, cases in which some supplementary agent is necessary, because too frequently in spite of minimal trauma, adhesions recur, particularly in those individuals with "adhesion diathesis" or "keloid tendency."

In a previous publication,[1] to which the reader is referred, a critique of the efficacy of the variously employed substances used to prevent the formation and reformation of adhesions is given. This report also contains the results of experimental observations concerning the effects of papain and trypsin in the prevention of the formation and reformation of peritoneal adhesions. These extraneous ferments were used for the purpose of supplementing a presumably insufficient amount of normal proteolytic enzyme. In a control group of animals adhesions reformed following their division in 100 per cent of instances. If saline solution was introduced into the peritoneal cavity following the division of adhesions, adhesions reformed in 86 per cent. If trypsin solution was introduced following the division of adhesions, they reformed in 57 per cent, whereas if papain solution was used, adhesions reformed in only 9 per cent. It was evident from this investigation that papain solution was more efficacious in preventing the reformation of adhesions following their division than either saline solution or trypsin. Although trypsin is a normal proteolytic ferment of the body and although it would seem that a normal ferment would be most efficient, the relatively poor results obtained by the use of trypsin solution are probably due to the fact that the tryptic activity was neutralized by antitrypsin which is normally found in the body. In corroboration of this Walton[2] showed that trypsin solution when placed in the peritoneal cavity rapidly loses its activity, but that papain remains active even after long periods of time and that its activity is not decreased by the addition of serum.

In addition to animal experiments parallel clinical observations have been made concerning the effect of papain in preventing serosal adhesions, and the present investigation is based upon an analysis of 231 cases, including the authors' cases, which have been treated by a total of 22 surgeons besides ourselves.* Observations by a large number of surgeons should be of more significance than those made by a small group. Although papain was definitely beneficial in the prevention of the reformation of experimentally produced adhesions following their division, it was realized that a prolonged period of observation of clinical cases so treated should be made before any definite conclusions could be drawn concerning its clinical value, because clinically it is not possible, as in the experimental animal, to relaparotomize patients at will in order to determine the presence or absence of adhesions, and because many patients can be perfectly comfortable with adhesions for years and then for no apparent reason develop signs of obstruction, which require reoperation. In the present clinical investigation, a subsequent laparotomy following the use of papain was performed in 37 cases. In most

* The papain used in the investigation was prepared in sterile form and generously supplied by the Parke-Davis Company.

instances the laparotomy was performed for some other lesion. An analysis of these cases is particularly valuable, because it is more comparable to the experimental observations.

In the 231 cases analyzed, the sex was stated in 169 cases, of which 142 (84.1 per cent) were females, and 27 (15.9 per cent) were males (Chart 1).

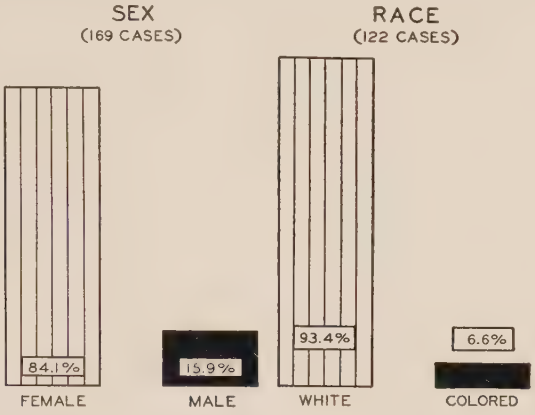

CHART 1.—Showing sex incidence in 169 cases in which it was stated.

CHART 2.—Showing race incidence in 122 cases in which it was indicated.

In 122 in which the race was indicated, 114 (93.4 per cent) were white and eight (6.6 per cent) were colored (Chart 2). In 140 cases in which the age was given, the youngest was four and the oldest 69. One (0.7 per cent) was in the first decade, 14 (10 per cent) were in the second decade, 39 (27.8 per

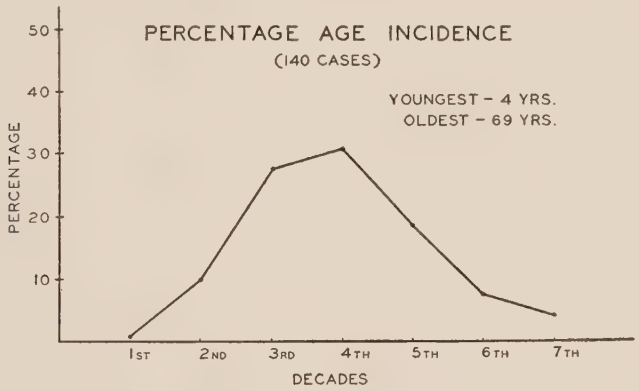

CHART 3.—Showing the age incidence according to decades in 140 cases in which the age was indicated.

cent) in the third decade, 43 (30.7 per cent) in the fourth decade, 26 (18.5 per cent) in the fifth decade, 11 (7.8 per cent) in the sixth decade, and six (4.2 per cent) in the seventh decade (Chart 3). Sixty per cent of the total number of cases were in the third and fourth decades.

In 122 patients in whom previous operations had been done, 317 operations were performed, or an average of two and one-half operations for each

patient. One hundred thirty-two (40.3 per cent) of the entire group of operations were for peritoneal adhesions and intestinal obstruction. As in 42 cases of the entire group the type of operation was not stated, the incidence of operation for adhesions and intestinal obstruction was 46.9 per cent in those in which it was stated. From the large number of operations previously performed for intestinal obstruction and adhesions, it is evident that many of these patients had an "adhesion diathesis." Fifty-seven (17.9 per cent of the entire group and 20.8 per cent of those in whom the type of operation was stated) had had a previous appendectomy. Forty-four (13.7 per cent of the entire group and 16.2 per cent of those in whom the type of operation was stated) had pelvic operations. Of these 44, eight had had a hysterectomy combined with other pelvic procedures, 25 had had some pelvic operation without a hysterectomy, and 11 had had cesarean section. Twenty-

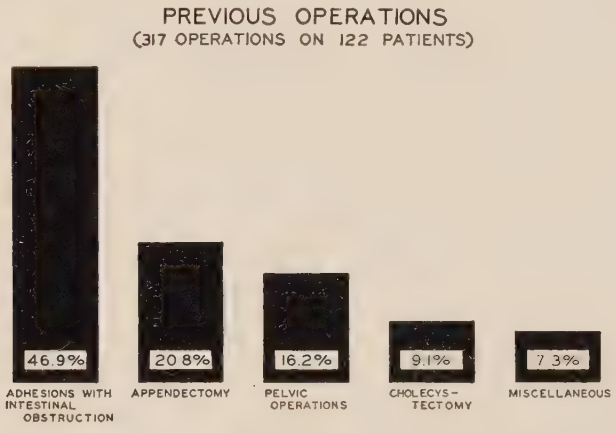

CHART 4.—Showing the percentage incidence of types of 317 operations performed on 122 patients in which papain was used.

five cases (8.8 per cent of the entire group and 9.1 per cent of the cases in which the operation was stated) had had cholecystectomy; two of these had had choledochotomies. Ten (3.1 per cent of the entire group) had had hernioplasties, six (1.8 per cent of the entire group) had had enterostomies, and one (0.3 per cent) had had an intestinal resection (Chart 4).

As mentioned above, a total of 317 operations was performed on 122 patients, an average of two and one-half operations for each patient. The greatest number of operations on one individual was 22, the smallest number was one. Fifty-five (45 per cent) had only one operation; 34 (27.8 per cent) two operations; ten (8.1 per cent) three operations; eight (6.5 per cent) four operations; five (4 per cent) five operations; three (2.4 per cent) six operations; two (1.6 per cent) eight operations; one (0.8 per cent) 18 operations, and one (0.8 per cent) 22 operations. Four per cent had nine or more operations; 12 per cent five or more; 26.6 per cent three or more, and 54.4 per cent two or more operations prior to the operation at which the papain was introduced into the peritoneal cavity (Chart 5).

Theoretically, papain solution might be used either for the prevention of the formation of adhesions at the original operation in an individual in whom there had been considerable trauma, but probably the best results and certainly the best evaluation is to be obtained in those cases in which adhesions have formed and in which the prevention of the reformation of adhesions

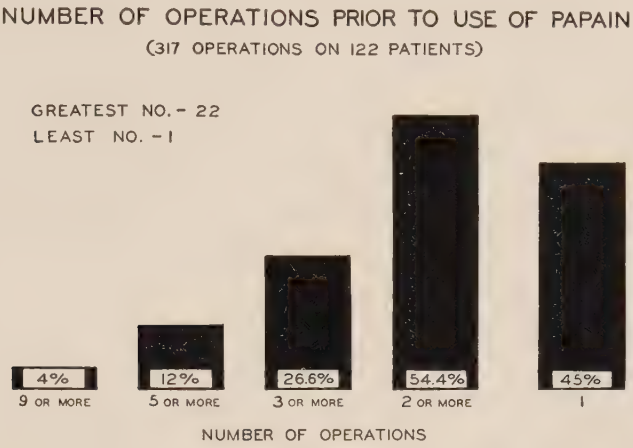

CHART 5.—Showing the number of previous operations performed on 122 patients.

following their division is desired. In the present clinical investigation, papain was used in 89 per cent of the cases to prevent the reformation of adhesions after their division. In only 11 per cent of the instances was papain used to prevent the formation of adhesions at the original operation (Chart 6).

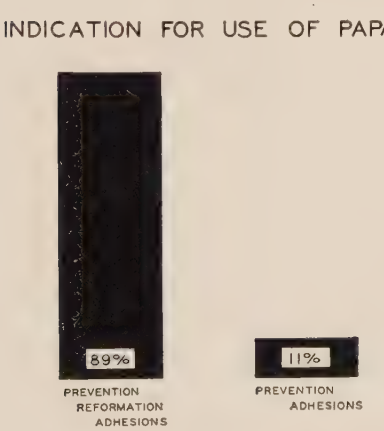

CHART 6.—Showing the indications for use of papain.

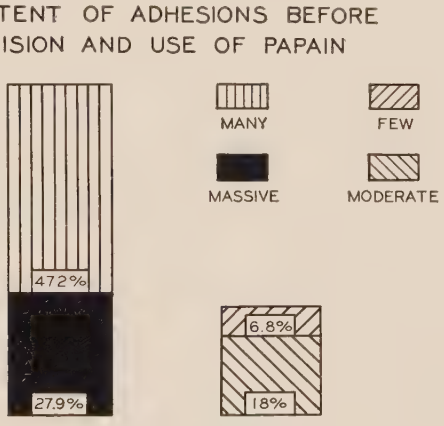

CHART 7.—Showing comparatively the extent of adhesions present before division and the use of papain.

In 161 of the observations a statement was made concerning the degree and the extent of the adhesions before their division at the time of operation. In 11 (6.8 per cent) the adhesions were few in number, in 29 (18 per cent)

they were moderate, giving a total of approximately 25 per cent in which the adhesions were not extensive before their division. In 76 (47.2 per cent) there were many adhesions and in 45 (27.9 per cent) the adhesions were massive, a total of approximately 75 per cent of extensive adhesions before their division (Chart 7).

In 109 cases collateral procedures besides the division of adhesions were performed. In all, 125 operations were done. Fifty-six (44.8 per cent) had pelvic operations, of which 44 (35.2 per cent) had pelvic operations without hysterectomy, and 12 (9.6 per cent) had pelvic operation combined with hysterectomy. Twenty-two (17.6 per cent) had an appendectomy; in 14 (11.2 per cent) a first stage of abdominal perineal resection of the rectum was done at the time of the papain introduction; in 12 (9.56 per cent) a cholecystectomy was done, and in 11 (8.8 per cent) a hernioplasty was done.

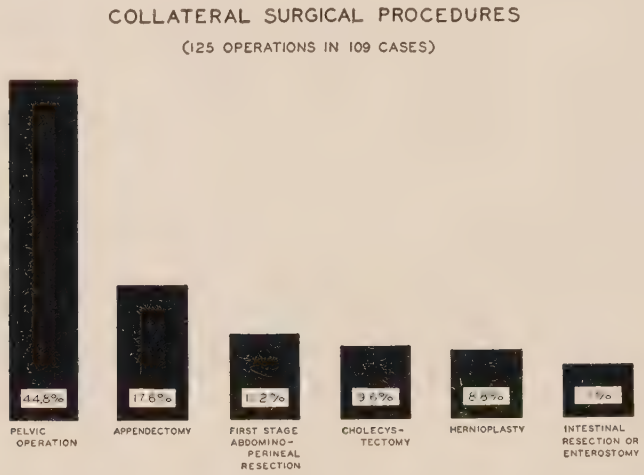

CHART 8.—Showing comparatively the types of the 125 collateral operations performed on patients at the time adhesions were divided and papain used.

In four (3.5 per cent) resection of the bowel and in four (3.5 per cent) enterostomy was done. In one resection of echinococcus cyst of the liver and in one cecopexy was done (Chart 8).

The period of observation after the division of adhesions and use of papain was stated in 190 cases. In 11 (5.7 per cent) it was between three and six months, in 17 (8.9 per cent) six months, in five (2.6 per cent) nine months, in 26 (13.6 per cent) one year, giving a total 17.2 per cent in which the observation was a year or less. In 21 (11 per cent) the observation was for one and one-half years, in 37 (19.4 per cent) for two years, in 21 (11 per cent) for two and one-half years, in 20 (10.5 per cent) for three years, in 14 (7.3 per cent) for three and one-half years, in 12 (6.3 per cent) for four years, in two (1 per cent) for four and one-half years, in three (1.5 per cent) for five years, and in one (0.5 per cent) for five and one-half years. Of the entire group, 9.3 per cent had been observed for four years or longer, 27.1 per cent for three years or longer, 57.5 per cent for two years

or longer, 82.1 per cent one year or longer, and only 17.2 per cent had been observed less than a year (Chart 9).

The mortality rate following the use of papain in the 231 cases in which it was used was 1.8 per cent, four deaths in the entire group (Chart 10). One of these patients died of acute gastric dilatation two weeks postoperatively. At postmortem no adhesions were found. In another patient death resulted from perforation of the bowel and inflammation of the ileum, and at postmortem many old adhesions were found. In the other two cases no clinical cause of death or autopsy findings were given.

In a total of 224 cases in which the results were given, in 186 (83 per cent) the results were classified as excellent. In an additional 13 (5.8 per cent) the results were classified as good, giving a total of 199 (88.8 per cent)

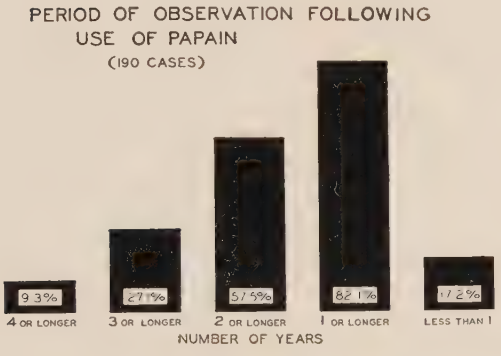

CHART 9.—Showing the percentage of patients who were observed for varying periods following the use of papain.

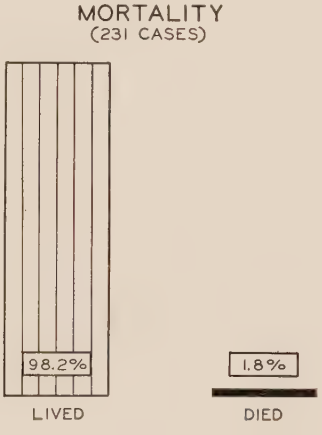

CHART 10.—Showing the mortality in cases in which papain was used.

as satisfactory results. In 17 (7.5 per cent) the results were classified as fair, and in eight (3.5 per cent) the results were classified as poor, giving a total of fair and poor in 11 per cent (Chart 11). The incidence of 88.8 per cent of satisfactory results is particularly interesting, because in the experimental investigation with papain performed approximately five years ago it was found that papain prevented the reformation of adhesions in 91 per cent of observations and that in only 9 per cent were there definite and dense adhesions present (Chart 12).

There were 37 cases in which subsequent operations were performed and in which the presence or absence of adhesions could be determined. Obviously the results in this group are more significant, because, even though a patient may be clinically free from symptoms, it is no indication that adhesions have not reformed; but the fact that 82 per cent of the patients had been observed for a year or longer and 57 per cent two years or longer and remained clinically well is a fairly good indication that probably most of them developed no adhesions. In the 37 cases in which a subsequent operation was done, largely for the removal of other viscera, in two (5.4 per cent) there was a reformation of many adhesions. In one of these the patient had

had an evisceration following the operation in which papain was used and which undoubtedly had a great deal to do with the reformation of the many adhesions. In 13 (35.1 per cent) there were some few adhesions present.

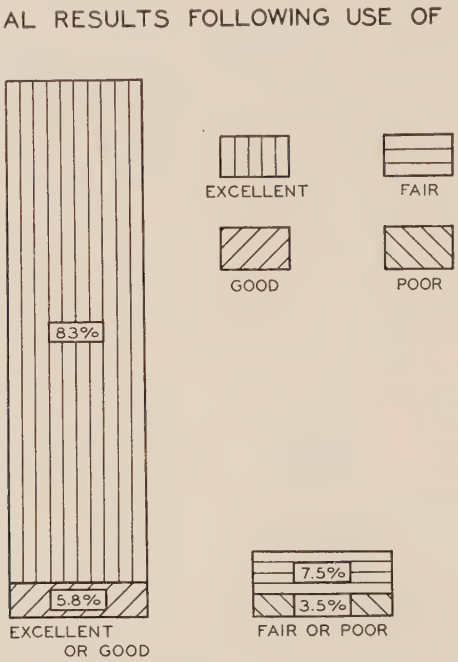

CHART 11.—Showing the incidence of the clinical results following the use of papain.

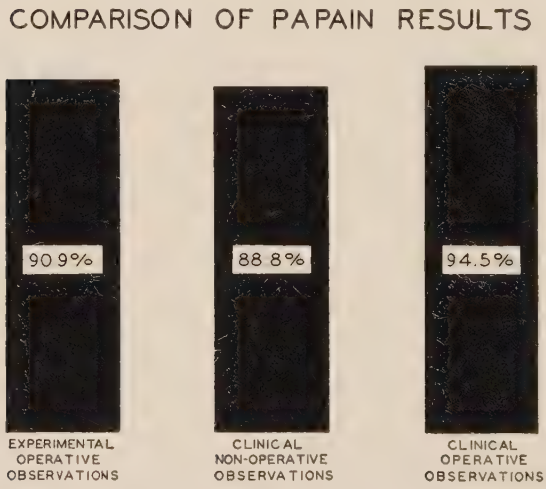

CHART 12.—Showing the findings at operations performed subsequent to the use of papain.

In ten of these (24 per cent of the whole) there was a statement made by the surgeon that the adhesions were considerably less than at the previous operation. In the three additional ones no statement was made as to whether

the adhesions were more or less than were found previously. In 22 of the entire group (59.4 per cent) there were no adhesions in the abdomen, although many and massive adhesions had been present prior to their division and to the use of papain. Thus, in 94.5 per cent papain was of distinct benefit in preventing or minimizing adhesions (Chart 13).

COMMENT.—Although it is extremely difficult to evaluate procedures in clinical cases and observation over a long period of time is necessary, the analysis of 231 cases studied in this investigation seems to justify the as-

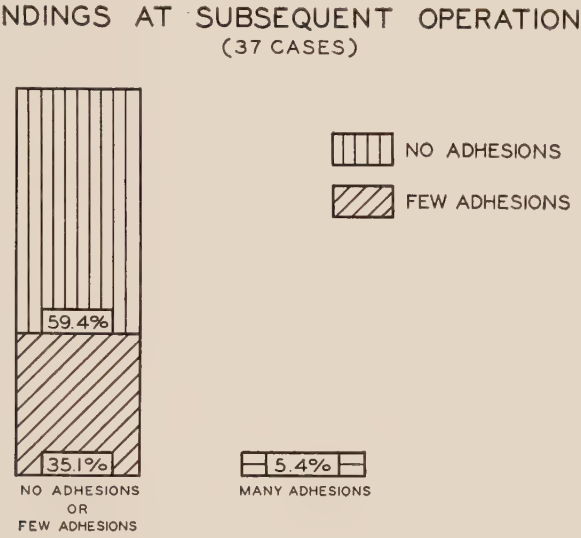

CHART 13.—Showing the close parallelism of satisfactory results observed in the experimental operative observations, the clinical nonoperative observations, and the clinical operative observations.

sumption that the introduction of papain solution into the peritoneal cavity following the division of adhesions is of value in preventing the reformation of adhesions. This is particularly evident when one considers that of 122 cases in which it was stated, 317 previous operations before the operation for the division of adhesions had been done, and especially because 46.9 per cent of these operations were for adhesions with intestinal obstruction, indicating that many of the patients were individuals with "keloid tendency" or "adhesion diathesis." The fact, too, that 54.4 per cent of these individuals had had two or more operations, 26.6 per cent had had three or more operations, and 12 per cent had had five or more operations supports this contention. In over three-fourths of the cases the adhesions at the time of the division were classified by the operating surgeon as many or massive. In addition to the division of the adhesions which in itself necessitated a certain amount of operative trauma and in many instances caused large areas of peritoneal denudation, collateral procedures were performed in 109 cases, which also added to the peritoneal trauma and predisposed to the reformation of adhesions.

Although many years must elapse before one can truly evaluate any substance as regards its ability to prevent the reformation of adhesions, it is

significant that in the present investigation, over one-half of the cases, 57.5 per cent, were observed two years or longer, over three-fourth of the cases (82.1 per cent) were observed one year or longer, over one-fourth (27.1 per cent) were observed three years or longer, and only 17.2 per cent were observed less than one year. Obviously a long period of observation and on a much larger group of cases is necessary before final conclusions concerning the value of papain can be drawn.

The low mortality rate, 1.8 per cent, in the entire group of 231 cases in which there was considerable operative manipulation, is an indication that the papain solution per se is harmless. In our own personal experience we have never observed any untoward results following its use, and we are convinced that it can be used without danger. The question has been raised a number of times whether papain should be used in the presence of infection, as in this way the digestion of the desirable and protective adhesions might be favored. Although the digestion of adhesions in the presence of infection is indeed undesirable, we believe that the use of papain in infection is harmless because the peritoneal trauma caused by the invasion of microörganisms lasts for a longer period of time than does the activity of the papain. In our own clinic we have limited the use of papain to those cases in which the prevention of the reformation of adhesions is desired and with few exceptions have not used it for the prevention of the formation of adhesions at original operation, because from an investigative standpoint the results obtained in the former group are more significant than those obtained in the latter group. We have limited the use of papain to the former, because one can never be certain that any individual who has had considerable intraperitoneal manipulation will develop adhesions. Of much more significance, however, is the prevention of the reformation of adhesions in the individual with adhesions which developed following an antecedent operation, because many of these patients have an "adhesion diathesis." If, however, papain is efficacious in preventing the reformation of adhesions, there is no reason why it should not be used in cases in which at the original operation there is considerable intraperitoneal trauma and in which the prevention of the reformation of adhesions is desired.

In our original investigations and in the earlier clinical cases, the papain was prepared, using saline as a diluent. Relatively recently, on the basis of additional experimental observations, we have been using Hartmann's combined physiologic solution as a diluent and believe that our results are better than those obtained with saline alone. Bogart[3] is of the opinion that a solution of 0.08 of 1 per cent of sodium citrate in distilled water is the diluent of choice. In our investigations citrate solution was not as efficacious as Hartmann's solution.

Whereas papain solution in the experimental animal is efficacious in dilutions as high as 1 to 40,000 and 1 to 50,000, in our recent clinical investigations we have been using dilutions of 1 to 20,000. As papain loses its efficacy very rapidly when in solution, it is imperative that the papain be kept in a dry form and that the solution be prepared immediately before its use. As

the sterile papain is supplied in 25 mg. ampules, it has been our custom to dissolve the contents of two ampules in 1,000 cc. of physiologic Hartmann's solution. This is done as follows: Fifty milligrams of papain are used for 1,000 cc. and prepared by emptying the 50 mg. of papain into a sterile mortar and adding a small amount of sterile Hartmann's solution. By the use of the mortar and a pestle the solution of the papain is greatly favored. A small amount of Hartmann's solution is then used to rinse out the vial in which it was originally contained and also to rinse out the mortar. After the division of all the peritoneal adhesions and just prior to putting in the last serosal suture, a catheter is introduced into the peritoneal cavity to which a funnel is attached. As much fluid is introduced into the peritoneal cavity as is possible. In the present series the amount varied from 250 to 1,500 cc., the average amount being 1,095 cc. In most instances a liter was introduced. Immediately after the introduction of as much of the papain solution as the peritoneal cavity will hold, the peritoneal suture is tied, keeping the solution in the cavity. Although theoretically there might be some danger of digestion of catgut suture material, we have not seen any such results.

Eighty-eight and eight-tenths per cent of excellent and good clinical results coincide well with results obtained by Bogart, who in 30 clinical cases had good results in 86.6 per cent. Our results are even more significant because in one of the eight in which poor results were obtained, the continuance of symptoms was probably due to the persistence of a chronic duodenal ileus, while in another instance, evisceration occurred following the use of the papain and possibly accounted for the production of many subsequent adhesions. In one of the 17 cases in which fair results were obtained, an echinococcus cyst of the liver was found at the original operation, and this may have been responsible for the lack of relief following the use of papain in that case.

Of greatest significance in this present study is the close correlation between the good results obtained in the experimental work done five years ago, the percentage of cases in which clinical relief followed the employment of papain, and the frequency with which an absence of adhesions was demonstrated at laparotomy subsequent to the use of papain. In the experimental animal, prevention of reformation of adhesions following their division was possible in 90.89 per cent. In the present investigation excellent or good clinical results were obtained following the division of adhesions and use of papain in 88.8 per cent. In the 37 cases in which a subsequent operation was performed following the division of adhesions and the introduction of papain, no adhesions were found in 59.4 per cent; a few adhesions were found in 35.1 per cent; in only 5.4 per cent (two cases) were the adhesions as great as they were before the use of papain and in one of these two cases evisceration had occurred following the operation and probably accounted for the recurrence of the massive adhesions (Charts 12 and 13). It would seem, therefore, that in approximately 94 per cent of instances papain had been efficacious in either preventing or definitely diminishing the number of adhesions. The fact that the results obtained with regard to the relief of

clinical manifestations and the demonstrated prevention of reformation or diminution in the number of adhesions in the reoperated cases corresponds so closely with the results obtained in the experimental animal as regards the prevention of the reformation of adhesions made these results more significant. It is of interest also that in Bogart's series good clinical results were obtained in 86.6 per cent of the cases.

SUMMARY

Two hundred thirty-one clinical cases are reported in which papain solution was used to prevent the reformation or initial formation of peritoneal adhesions. Many of these patients were considered to have a "keloid tendency" or "adhesion diathesis," because 122 patients in the group had had previous operations and in 46.9 per cent of instances in which the type of operation was stated, it had been for adhesions and intestinal obstruction. The period of observation following the division of adhesions and use of papain was one year or longer in 82.1 per cent. In a total of 224 patients in which the clinical results were given the results were classified as excellent in 186 (83 per cent) and in an additional 13 (5.8 per cent) the results were classified as good, giving a total of 199 cases (88.8 per cent) in which satisfactory results were observed. In 17 cases (7.5 per cent) the results were classified as fair and in eight (3.5 per cent) the results were classified as poor, giving a total of fair and poor results in 11 per cent.

Thirty-seven cases were relaparotomized following the use of papain. In 22 (59.4 per cent) of these there were no adhesions in the abdomen although many and massive adhesions had been present prior to their division and to the use of papain; in 13 (35.1 per cent) there were some few adhesions present, but many less than before the use of papain, whereas in two (5.4 per cent) there was a reformation of many adhesions. There is a close correlation between the clinical results (88.8 per cent satisfactory), the observations made at operations subsequent to the use of papain (94.5 per cent satisfactory results), and the experimental observations (90.9 per cent satisfactory results) done five years ago. None of the four deaths (1.8 per cent) which occurred in this series of 231 cases could be attributed to the use of the papain solution.

NOTE.—We wish to express our appreciation of the following men who have assisted in this clinical investigation by furnishing reports of cases in which they have used papain for the prevention of peritoneal adhesions: Charles H. Arnold, William L. Bendel, W. E. Bird, Leon M. Bogart, Aza W. Collins, John B. Deaver, John F. Denton, A. R. Dickson, James Q. Graves, Donald Guthrie, E. S. Hicks, W. Kernan Irwin, Walter C. Jones, Morris Joseph, Joseph E. J. King, George V. Lewis, S. Mirabella, Damon P. Pfeiffer, A. A. Skemp, John G. Snelling, Benjamin W. Ward, and George W. Wright.

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DELAYED WOUND HEALING FOLLOWING NEPHRECTOMY FOR TUBERCULOSIS

ANALYSIS OF CAUSE

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NEW YORK, N. Y.

IT IS a well known fact that the lumbar incision used in the removal of a tuberculous kidney in a certain proportion of cases heals less rapidly than nephrectomy wounds in which the kidney shows no evidence of tuberculosis. The disturbance in the wound healing in these tuberculous cases varies from an extensive breaking down of the apparently healed wound to the formation of one or more fistulae. The cause of this peculiar behavior of the wounds is apparently a local tuberculous condition, though in only a small percentage of the cases does the granulation tissue removed from these wounds show typical tubercle formation.

To date, no one has found a method of preventing this complication which may lead to great inconvenience for the patient occasioned by prolonged discharge and more or less incapacitation. Some of these wounds remain open and discharge for years, so that the individuals are under the doctor's care for a very long time. The exact cause of this type of wound infection has as yet not been recognized, though one of us[1] has repeatedly called attention to the possibility that the lumbar muscular wound becomes infected by tubercle bacilli, which have been thrown into the circulation by the manipulations incidental to the nephrectomy. An infrequent cause of such wound infections is the gross contamination of the wound by spilling, which, however, does not regularly produce the typical slow healing lumbar wounds. The usual explanation found in the literature for these slowly healing wounds attributes them to the remaining tuberculous ureter and generally these infected wounds and tuberculous ureter fistulae are considered synonymous.

The object of this paper is to call attention to the fact that in a large series of cases we have been able to show definitely that the diseased ureter has nothing to do with the slow healing and the tuberculous infection of the muscular wound, but that the essential underlying causation is a traumatic bacteriemia induced by the operator, which leads to a deposition of tubercle bacilli in the vascular muscle wound and in the traumatized deeper tissues. Only occasionally is the wound infection due to gross spilling of pus containing tubercle bacilli, the result of an accidental rupture either of an abscess in a calix, of the pelvis or of the ureter during the delivery of the tuberculous kidney. In a study of 281 cases, we have found no evidence of tuberculosis of the perirenal fat, such as has been described by Legueu, except in the sclerosed perinephric fat, which is adherent to the kidney in long standing

cases. In the early cases, before the perinephric fat has been grossly involved, we have, as yet, never detected evidence of tuberculosis in the microscopic study of this tissue, and therefore cannot consider it an underlying cause of the unfortunate accident and clinical picture.

For many years I (E. B.), have been deeply interested in the problem of bacteriemia caused by trauma to the patient from within, or from without, by instruments or operations. Cases have been observed in which the passage of biliary and kidney calculi have been associated with chills, a sharp rise in temperature and transitory bacteriemia. Similar cases have been seen after urethral or ureteral instrumentation. Other writers, including Winter, Schottmueller, Buschke, Dudgeon, Mitchener, etc., have called attention to traumatic bacteriemia. More recently Seifert, in 1925, reviewed most of the work and found that operations in an infected terrain frequently led to bacteriemias, especially in early infections and in vascular areas and that the rougher the manipulations and the longer the operation, the more frequent were the bacteriemias. In Staphylococcus aureus and albus infections, such bacteriemias developed in 54 per cent of 55 cases; in streptococcus (erysipelitus) infections, bacteriemia developed in 30 per cent of 11 cases; in colon bacillus infections, bacteriemia developed in 25 per cent of 11 cases. Interesting to relate, he found in non-infected patients in aseptic operative fields (*e.g.*, goiter operations) transitory, postoperative bacteriemias. The clinical observation has been made by many that following operations upon tuberculous foci, especially in tuberculosis of the generative organs and the kidneys, occasionally a diffuse miliary tuberculosis develops with tuberculous meningitis as the outstanding symptom. The postoperative course of many of these cases is accompanied by spiking temperatures typical of a sepsis and some, perhaps the majority, gradually become normal, whereas the others die, having developed the definite and fatal signs of tuberculous dissemination. In the first group, apparently the bacteriemia was transitory, as is frequently the case with the coccal and colon group.

Experience has shown that it is particularly difficult to pick up the tubercle bacillus in the blood stream. In well over 30 patients suffering from renal tuberculosis, we (E. B., and the late Dr. Eugene Bernstein) injected the blood taken just before and just after the nephrectomy into guinea-pigs, and in three cases were able to produce tuberculosis. Graf called attention to tubercle bacilli in the blood following operations upon tuberculous foci. Seifert emphasizes the great difficulty of this research, which is explained by A. Calmette by the fact that blood or serum is liable to be toxic to guinea-pigs in amounts equal to 8 cc. In view of the postoperative incidence of miliary tuberculosis following nephrectomy of a tuberculous kidney, J. Israel suggested that the trauma of the operation probably led to dissemination of the bacilli. Zuckerkandl called attention to a similar dissemination after curettage of a tuberculous wound. H. Wildbolz and A. Westerborn called attention to the dissemination and production of miliary tuberculosis by cys-

toscopy and Kearns reported similar dissemination following retrograde pyelography.

Although the clinical evidence of a sepsis due to traumatic dissemination of tubercle bacilli during operations is overwhelming, the work of Eugene Bernstein on the inoculation of guinea-pigs with the blood of patients before and after nephrectomy for tuberculosis is, as far as I can gather from the literature, the outstanding laboratory evidence of the phenomenon under discussion.

There is rather general agreement as to the frequency of disturbed healing in the lumbar wound in all clinics where many cases of tuberculosis come to nephrectomy. Israel in the study of his material and other clinics found 29.4 per cent fistulae still present after six months. Braasch found 43 per cent healed within three months and 87 per cent within one year of operation. Wildbolz in 175 nephrectomies found 44 fistulae, or approximately 25 per cent. In our series of 281 cases, it is imperative that we divide our cases into two groups, those in whom a simple nephrectomy (lumbar) was performed, and those in whom an aseptic nephro-ureterectomy[2] was effected through two incisions, the typical posterior vascular muscle incision, and the anterior avascular rectus sheath extraperitoneal approach to the ureter. In the first group, there were 243 nephrectomies with 58 wound sinuses or fistulae (23.8 per cent), whereas in the second group there were 38 aseptic nephro-ureterectomies and nine (23.7 per cent) developed lumbar wound sinuses, whereas only three cases developed mild infections in the rectus sheath incision. In one of these the infection of the anterior incision seems to have developed from an ureter stump abscess 26 days following the operation, the wound having closed before this abscess developed. In the second there was gross spilling of infected pus on cutting through the pelvic ureter between the ligatures and in the third case the pelvic ureter was considerably traumatized, as it was very adherent.

In both series of cases the incidence of trouble in the lumbar wound was the same, whether the ureter was removed or not. This observation makes it very clear that the generally accepted explanation, that these lumbar wound fistulae are caused by the tuberculous ureter, is untenable. Moreover, the striking difference between the behavior of the two wounds, the lumbar and the rectus, despite the presence of the tuberculous ureter stump in the anterior, avascular, rectus wound, together with the other evidence submitted, points to but one conclusion; namely, that traumatic, tuberculous bacillemia is induced by the operator, and leads to infection and tuberculosis of the rather vascular posterior wound, while the anterior wound, being practically completely avascular, is spared.

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Discussion.—Dr. Hugh Cabot (Rochester, Minn.). Doctor Beer referred to the occasional occurrence of miliary tuberculosis. It is a rare complication, but, as he suggests, does occur. One frequently sees (I have no doubt he will recognize them) sharp reactions following nephrectomy. They are characterized by a rise of temperature out of proportion to the rise in pulse, no other symptoms, and no evidence of ordinary infection. I have long regarded them as tuberculin reactions, though I am by no means sure that they are caused only by the toxins and that they are not in fact due to the massage that is inevitable in removing the kidney, and thus to the presence of tubercle bacilli in the circulation. I think this reaction without other infection makes it clear that it is something related to the tuberculous process.

My experience leads me to suggest that patients operated upon in, what we are pleased to call, the early stages of the disease (of course one never sees the early stages of renal tuberculosis; we always see the late stages), in the stage of moderate ulceration of the papilla, often do very badly. Possibly this may be related to the lack of protection which the patient receives from the more chronic process. I do not suggest that we allow a tuberculous kidney to remain until the bladder has become thoroughly infected and damaged, but I do feel that operation upon, what we are pleased to call, the early stages of the disease is not always wise. There is a certain amount of protection developed as the result of the longer development of the lesion.

My own opinion is that most of the persistent sinuses are related to residual tuberculosis, partly in the remaining capsule, and, in the more chronic cases, partly to actual tuberculous nodes lying in the neighborhood of the pedicle. I have had several cases in which I was aware that I had left tuberculous disease, and those patients have developed sinuses. Finally, my own experience over a good many years with the routine use of tuberculin after these operations suggests that it has reduced the incidence of sinus formation more than half.

I should hesitate to adopt, what I take to be, Doctor Beer's suggestion that we abandon the lumbar route and use an anterior approach to these kidneys. I am not at all sure that we should, in fact, remedy the difficulty by that method.

THE RÔLE OF SURGERY IN THE TREATMENT OF ACTINOMYCOSIS

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This presentation is in essence a brief recital of the writer's personal experience with the treatment of actinomycosis. The mode of origin of this disease is still an enigma, in that the organisms which are found in the diseased tissues of patients or animals having actinomycosis have not been found elsewhere in nature or even in these same hosts when free from the disease; the causative organism can be grown, to be sure, on artificial culture media. Actinomycosis-like organisms inhabit the mouths of healthy individuals, but in the main they are believed not to be pathogenic. The organism found almost invariably in human cases of actinomycosis is the anaerobic *Actinomyces bovis* which also causes "lumpy-jaw" in cattle. When the mystery of how this organism reaches and sets in motion the series of events which leads to actinomycosis is solved, intelligent prophylactic measures may be devised which may spare men the sufferings and ravages of the disease.

Sites Where Actinomycosis May Occur.—There are essentially three regions in the body where actinomycosis commonly occurs, *viz.*, (1) the head and neck, (2) the thorax, and (3) the abdomen. The usual portal of entry for cervicofacial actinomycosis is believed to be the mouth; for thoracic actinomycosis, aspirated or ingested organisms which lodge in pulmonary tissue or penetrate the esophagus; the usual lodgment of organisms which give rise to abdominal actinomycosis is the ileocecal segment of the intestinal canal.

Pathology.—Lodgment of the ray fungus, *Actinomyces bovis,* in tissue brings about a granulomatous type of reaction in which the features of an acute as well as chronic infection may be concurrently noted. Immediately about the organism a zone of cellular activity usually occurs in which are present large masses of polymorphonuclear and mononuclear leukocytes, epithelioid cells, and a rich network of young blood vessels. This reaction becomes manifest to the unaided eye as abscess formation, with burrowing pus channels filled with purulent collections containing the yellow, sulphur-like granules of the *Actinomyces* colonies. The vascularity of this granulomatous process is always striking at operation. Hemorrhage from this tissue when curetted away may be alarming, lending the impression that a large blood vessel may have been opened, yet, slight gentle pressure with a gauze pack usually serves to arrest it. Randall, who studied the cases of actinomycosis observed at this hospital up until 1933, points out that the

yellow color of the exudate is due in large part to the presence of pseudoxanthoma cells rich in lipoids. The *Actinomyces* are nonmobile, but are carried into the surrounding tissues by the macrophages.

Peripheral to this area of necrosis and liquefaction is observed a proliferation of dense connective tissue—an attempt to localize and stop the destructive process. The brawny induration observed clinically in cases of actinomycosis is afforded by this keloid-like proliferation of connective tissue. Central softening and peripheral induration with the suggestion of remarkable vascularity, as imparted by the color of the skin, are the gross features of the actinomycotic process which is about to rupture through the skin. Spontaneous fistulization with discharge of yellowish exudate is not unusual. In man, primary involvement of bone is infrequent and is usually observed as an extension from an adjacent process; in cattle, involvement of the jaw bone is the most frequent initial lesion of true actinomycosis.

The Spread of Actinomycosis.—When the disease is recognizable clinically, the portal of entry ordinarily remains but a conjecture. The proximity of the diseased process to one of the commonly accepted sites of origin of the disease merely suggests it as the portal through which the infection invaded the adjacent structures. Actinomycosis exhibits an extraordinary ability to extend into healthy tissue, leaving no trace of the disease at the site of entry. This is particularly true of abdominal actinomycosis. It is likely that the unexpected finding of isolated actinomycosis in a subphrenic abscess, in the kidney, in the urinary bladder, the female generative organs, in the abdominal wall, or a collection of actinomycotic exudate eroding the lower thoracic or lumbar spine commonly have their origin in actinomycosis which initially found lodgment in the cecum.

Actinomycosis rarely becomes generalized in the sense in which a tumor metastasizes, though instances apparently have, however, occurred to indicate that distal spread by the blood stream does take place. Most of such cases concern the invasion of a pulmonary vein by an actinomycotic process in the lung: its entry into the left heart and its propagation as a thrombus into one of the cerebral vessels, with lodgment in the brain. Jacoby[21] has reviewed a number of such instances. These cerebral actinomycotic abscesses are sometimes multiple. Similar, but less frequent, spread to liver, spleen, or kidney has also been noted. It is particularly the thoracic and abdominal varieties of actinomycosis which give rise to blood stream invasion. Because of the remarkable invasive features of actinomycosis by which the disease spreads by direct extension, involvement of liver and spleen may occur in either abdominal or thoracic actinomycosis by direct migration without the agency of blood stream metastasis.

There is a fairly large number of instances of isolated actinomycosis of the urinary bladder, the female generative organs, the kidney, and even bone, the origin of which remain uncertain. As was suggested above, undoubtedly some of these may have their origin in so called abdominal actinomycosis,

presumably the residual of an actinomycotic process which migrated out from the intestinal canal.

That actinomycosis may have reached these organs by direct spread from an actinomycotic focus which had its origin elsewhere than in the intestinal canal is a possibility. The instances of isolated actinomycosis of the stomach which have been observed undoubtedly have their origin in the ingestion and direct lodgment of the *Actinomyces* in the stomach.

Extension from cervicofacial actinomycosis may occur into the thorax by way of the supraclavicular fossa; invasion of the cranium through the orbit or by way of the foramina at the base of the skull or by direct erosion of bone have all been observed. The importance of early recognition and adequate treatment of the lesion to obviate these unfortunate occurrences is immediately apparent.

Diagnosis.—That actinomycosis of the thorax or abdomen may not be early identified is obvious; why actinomycosis of the cheek or neck should not be recognized early is difficult to understand. A lesion which may be seen and palpated should suggest invoking aids to affirm or deny the presence of actinomycosis. Mere consideration of the possibility of the lesion goes a long way toward its identification if every such lead is pursued to its logical conclusion.

The determination of the actual presence of actinomycosis rests upon bacteriologic means. The exudate recovered from an area of softening by needle aspiration or that obtained from a sinus or recovered by curettement at operation is put out on gauze or placed in water where the granules may be more readily identified. Suspicious looking granules are placed on a glass slide in a drop of strong potassium hydroxide and this wet preparation is examined immediately. The sulphur-like granule of an actinomycotic colony thus examined presents the appearance shown in Fig. 1A. A coiled group of intertwined mycelial threads terminating in clubs is the typical picture. The *Actinomyces bovis* is anaerobic and gram-positive. The *Actinomyces*-like organisms in the mouth are usually aerobic. Dr. A. T. Henrici,[17] Professor of Bacteriology at the University of Minnesota, who has examined the exudate from the cases of actinomycosis observed at the University Hospital, tells me that *Actinomyces bovis* has been the organism uniformly found.

In instances in which fistulization has already occurred and drainage of exudate is slight, curettement of the sinus followed by paraffin embedding and staining with hematoxylin and eosin of a cut section will usually succeed in demonstrating the organisms when they cannot be identified in the exudate (Figs. 1B, 1C). When sinuses have long been present in actinomycosis, the organism may be difficult to find. They are most readily found in the lesion which has yet not been drained.

Differential Diagnosis: *Cervicofacial Actinomycosis.*—Very few of the cases which have come to the University Hospital for treatment have been correctly identified before admission. Persons of middle age, who have

FIG. 1.—(A) A sulphur granule (Gram's stain ×400); this is the typical ray fungus rosette. The conformation of the twisted mycelial threads is shown. (B) A number of *Actinomyces* colonies surrounded by leukocytes. (C) A Gram's stain of a sulphur granule showing the intertwining mycelial threads of the *Streptothrix* (×400).

the disease, are usually sent in with the diagnosis of malignancy; in younger persons the most frequent diagnosis is tuberculosis of the lymph nodes. The trismus of the muscles of the jaw and the induration of the cheek and neck with discharging sinuses frequently do suggest the picture of a late intra-oral malignancy. If the mouth can be opened adequately for examination, the absence of an ulcerating lesion should at once give increased credence to the belief that the lesion is probably actinomycosis. Malignancy, tuberculous lymph nodes, osteomyelitis of the jaw, and suppuration in a branchial cyst may mimic the appearance of this variety of actinomycosis. The finding of the organism establishes the nature of the lesion.

FIG. 2.—Result after complete excision of an extensive actinomycosis of the right cheek (Table I, Case 7). The trismus which patient had quickly disappeared. Cosmetic and functional result is satisfactory. Subsequent experience has shown that curetting away the dead tissue alone usually suffices to bring about the same result; it does, however, take a somewhat longer time to bring about complete healing.

Thoracic Actinomycosis.—Early recognition of the lesion is likely to be accidental. In my own experience, it is the occurrence of an empyema which is threatening to rupture through the thorax in the vicinity of the breast on the anterior chest wall which gives the first suspicion of the presence of thoracic actinomycosis. Kirklin and Hefke have indicated that the roentgenologist may occasionally diagnose the lesion when the lung, pleura, and chest wall are involved—that is, an intrapulmonary lesion which simultaneously gives rise to a periostitis should suggest actinomycosis. My experience has been that the clinician will usually suspect the presence of the lesion before

the roentgenologist will be able to suggest the possibility of its presence from roentgenograms of the chest. An instance in which, after surgical drainage of an empyema, the true nature of the lesion remained still undiscovered for a long time, has come to my attention (Table II, Case 7). The free bleeding which followed probing of the tract at once suggested the possibility of actinomycosis. Search disclosed the presence of the fungus. Pulmonary abscess, bronchiectasis with abscess or carnification of the lung, tuberculosis, malignancy of the lung or pleura, pyogenic empyema, unresolved pneumonia, syphilis of the lung, blastomycosis, and hydatid cyst may give rise to confusion.

Abdominal Actinomycosis.—Most instances of abdominal actinomycosis are operated upon for appendicitis. Exudate may be found around the appendix, the true nature of the lesion remaining wholly unsuspected. An

FIG. 3.—Actinomycosis in a child of 13 (Table I, Case 11). After two curettements complete healing resulted. Final result.

intestinal fistula may form or an abscess may appear in the incision subsequent to complete healing. The late occurrence of a subhepatic, subdiaphragmatic or perirenal abscess necessitating drainage may be the means by which the process is identified. Any sinus developing spontaneously in the abdominal wall should be looked upon with suspicion. Carcinoma, particularly in the cecum; Hodgkin's disease; appendiceal abscess, and its complications; tuberculosis of the bowel and peritoneum; so called regional ileitis with abscess formation; perinephritic abscess; and chronic granulomata are the ordinary conditions with which abdominal actinomycosis may be confused. Shiota relates that localized actinomycosis has been cured by appendectomy or intestinal resection. Cure after gastric resection, in which a surprise finding of actinomycosis was found histologically, has been reported in a few instances. Similar cures have attended drainage of a subdiaphragmatic or pelvic abscess, actinomycotic in nature, which in all likelihood had its origin

in an ileocecal lesion; like results have attended excision of an actinomycotic kidney, as well as excision of an abscess of the fallopian tubes or ovaries or a process in the uterus which was found to be actinomycotic.

Treatment.—Actinomycosis has been known as a disease entity affecting man for almost 60 years. During this time a large number of agents have been recommended and tried in combating the disease. Only three have enjoyed wide usage, *viz.*, surgery, potassium iodide, and irradiation. Among other remedies less frequently employed, the following may be enumerated: vaccine, methylene blue, copper sulphate, neoarsphenamine, and foreign protein injections.

Roentgen therapy of actinomycosis, first employed by Harsha of Chicago, did not gain many adherents until after Levy of Breslau again advised its use in 1913. Its endorsement by Heyerdahl[18] of Oslo and New and Figi in this country have led to its wide employment in the treatment of this condition. That the method has virtue is adequately attested to in the numerous papers extolling the remedial properties of this therapeutic agent. The manner in which it operates is not clear. Kleesatel and Ingber[20] have both indicated that the roentgen ray or radium has no effect upon the organism.

The employment of potassium iodide in the treatment of human actinomycosis originated from veterinary medicine (1892). Veterinarians have since learned that potassium iodide is of no value in the treatment of *Actinomycosis bovis.* In actinobacillosis, a disease of cattle and swine which mimics the pathologic aspects of true *Actinomyces bovis* closely, potassium iodide acts as a specific, terminating the disease. Actinobacillosis in man, on the contrary, has only rarely been observed. That the administration of iodide in any granulomatous infection may bring about some improvement is a matter of common knowledge. It is, however, to be acknowledged that potassium iodide is in no sense a specific in the treatment of actinomycosis, and it may also be said that it does not warrant the reliance and confidence generally accorded it. Many patients with actinomycosis are given potassium iodide until symptoms of iodism with profound listlessness and loss of appetite supervene.

An experience of many years with the employment of potassium iodide, irradiation, and surgery in the treatment of actinomycosis leads the writer to believe that surgery is the agent of greatest worth. One cannot read the papers of Heyerdahl and Engelstad[11, 12] and escape the impression that irradiation has real merit. Experience with cases treated under my supervision and cases treated elsewhere, which have later come to my attention, have convinced me that surgery is the most direct therapeutic measure with which to attack the disease. As has been stated above, *Actinomyces bovis,* which is the organism responsible for most cases of actinomycosis observed in man, is an anaerobe. In the débris of the dead tissue which the diseased process brings about, in which the oxygen tension is zero, the organism thrives and is carried off by the macrophages into the healthy tissue in their attempt to combat the disease. Here again, new abscesses develop with death of more

FIG. 4.—(A) Extensive actinomycosis in a child of seven (Table I, Case 13), photograph made on day of operation (9/29/34). The patient is trying his best to open his mouth. In addition to the trismus there is also slight weakness of the facial nerve. (B) Photograph taken 20 days after (A) (10/18/34). One curettement has been done in the meanwhile. The trismus has now disappeared. (C) Taken at the same time as (B); the plain small gauze packing is shown in the small incision made at the time of operation through which the curette was introduced. These gauze packs are changed as occasion demands (once a day to once in two or three days). The wounds healed following two curettements. (D) A recent photograph.

tissue and the establishment of new areas of low oxygen tension which constitute favorable culture media for the perpetuation of the disease. What is obviously needed is the excision of these areas of dead and dying tissue which propagate the disease. Why not employ the most direct approach known in trying to thwart the destruction of this disease? What wonder that the disease may extend itself while the indirect agents of potassium iodide and irradiation alone are being used. Yet, some roentgenologists, familiar only with the results of the agent which they employ, would relegate surgery in the treatment of actinomycosis to a place of subsidiary import or eliminate it altogether.

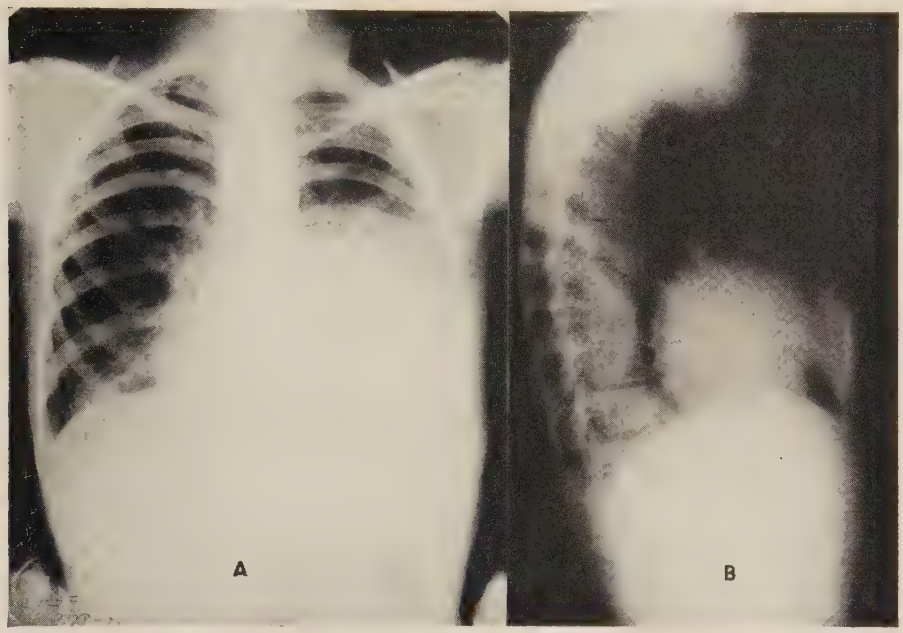

FIG. 5.—(A) Postero-anterior roentgenogram of a patient with thoracic actinomycosis, at time of admission, February, 1931 (Table II, Case 2). (B) Left lateral exposure. Showing mass in the left lung.

Having had the opportunity to observe the tardy and disappointing effects of a combination of potassium iodide, irradiation, and surgical restraint in the treatment of extensive cervicofacial actinomycosis, I resolved to test the efficiency of these therapeutic agents by using them separately. The results of energetic surgical treatment alone have been so gratifying that at the University Hospital we have come to rely solely on this measure of relief in the treatment of actinomycosis. At first, excision of the diseased tissue was practiced, but soon I learned that curetting away the dead tissue, leaving the wound open and providing drainage is all that is ordinarily necessary. I have been amazed to see how quickly traces of the disease will disappear and the wound heal with removal of the dead tissue. Two or three curettements alone have sufficed to successfully terminate extensive cases. In a recent instance of cervicofacial actinomycosis which had been treated else-

where by incision, large doses of potassium iodide, and skillfully administered irradiation, seven months of treatment had only led to the impression that the havoc of the disease was beyond repair (Table I, Case 14). Repeated roentgen ray treatments and continuous administration of potassium iodide had failed to stop extension of the disease. Weight loss, weakness, languor, and apathy were striking. At the first séance, the soft discolored abscess-ridden tissue of the entire left and a part of the right side of the neck were readily scraped away with the curette. The wound was packed with gauze to control hemorrhage. Two subsequent limited curettings brought about complete healing. In four months' time, less than a month of which was

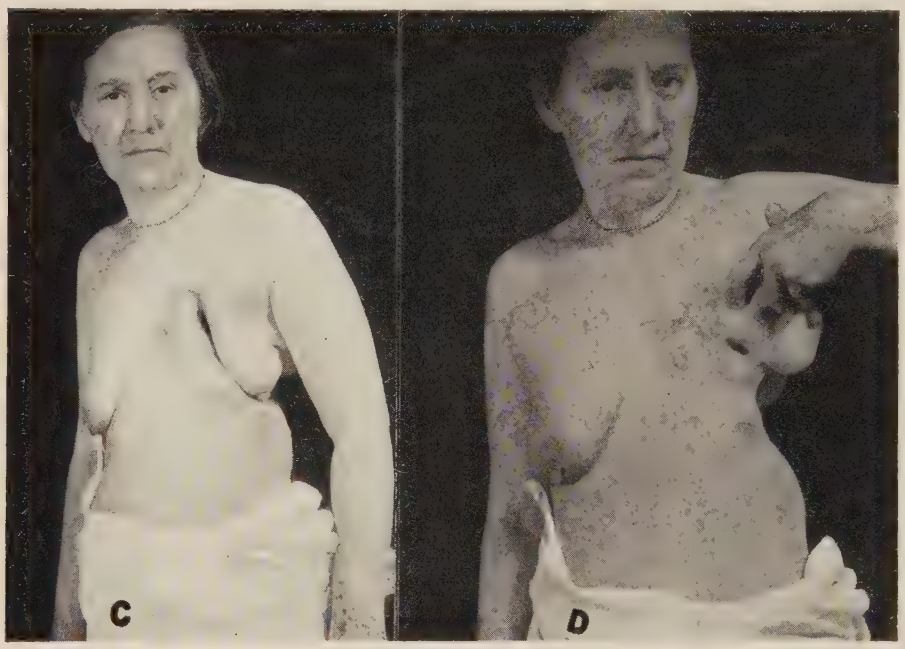

FIG. 5.—(C and D) Photographs made November 21, 1934. No evidence of actinomycosis had been found in last 18 months on three separate hospital admissions. Patient died, however, of a brain abscess March 25, 1935; whether this abscess was actinomycotic in nature is not known.

spent in the hospital, the entire raw area had granulated and become epithelialized and the wound completely healed, all without further recourse to the therapeutic agencies of potassium iodide and irradiation.

Another case was admitted in a moribund condition a few years ago to the University Hospital, as result of the ravages of a cervicofacial actinomycosis, which had been treated over an eight year period largely by potassium iodide and irradiation and an occasional incision (Table I, Case 2). A more direct approach, by energetic removal of the diseased tissue by curettement probably would have influenced the diseased process favorably.

There were two other fatal cases in the cervicofacial group of actinomycosis observed at the University Hospital. One of these (Table I, Case 1) was treated in 1925 by large doses of potassium iodide, irradiation, and roentgen therapy. The only surgery employed was the occasional opening

of an abscess by incision. It was really the disappointing experience with the remedial agents of potassium iodide and irradiation in this case which led to the employment of more aggressive surgery. Since 1930, because of the quick response obtained in energetic surgical removal of the dead tissue in actinomycosis, I have come to rely on surgery alone and have abandoned the use of potassium iodide and irradiation.

Case 3 in Table I had an extensive actinomycosis of the scalp, neck, and face of three years' duration when first seen. He was observed and treated at the University Hospital over a period of about a year and one-half. With the employment of multiple incisions and curettement, the lesions in the soft tissues cleared up fairly rapidly. A small lesion persisted near the right auditory meatus and another near the canthus of the right eye. He eventually

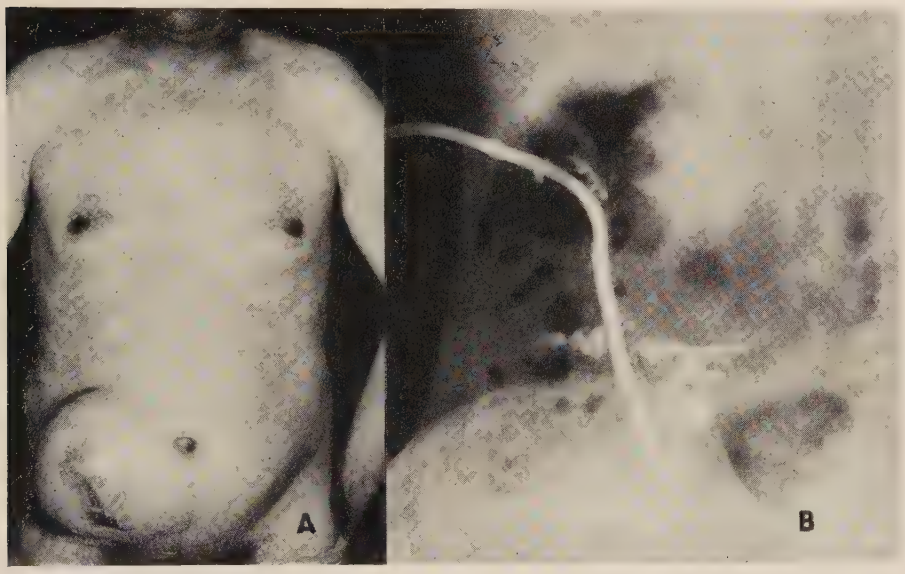

FIG. 6.—(A) Patient with abdominal actinomycosis (Table III, Case 4). The scar and hernia from an antecedent appendectomy for suppurative appendicitis are shown. Two curettements brought about closure of the sinuses, which however have opened up occasionally. (B) Lipiodol injection of sinus tract before curettements. No communication with the bowel is apparent.

developed signs of meningitis, from which he died. Exenteration of the contents of the orbit and opening up of the auditory canal earlier probably would have avoided this unfortunate outcome. At the time, however, it was hoped that less radical measures would suffice. Additional employment of potassium iodide and irradiation failed to do what more energetic surgical intervention probably could have accomplished.

My own experience with the treatment of thoracic and abdominal actinomycosis affords no occasion for optimism. Yet, if the disease can be diagnosed early and prompt evacuation of the dead tissue effected, the means of extending the disease is done away with. I believe that at least some of these cases are amenable to treatment. The literature is most pessimistic over the outlook in thoracic actinomycosis, and the prognosis in the abdominal variety does not appear much brighter. Yet, cures in thoracic as well as

RESULTS OF TREATMENT OF ACTINOMYCOSIS*

Table I. Cervicofacial Cases

Patient, Hospital Number, Sex, Age, Admission Date	Extent of Lesion	Treatment	Result	Remarks
Fatal cases:				
(1) A. S.—33878 Male—43 yrs. 11/16/25	Right side face and neck; left side face and neck	Roentgen ray, radium, KI, incisions	Died 7/19/26	Energetic sugrical intervention (curetting) probably would have cured.
(2) M. F.—621288 Female—31 yrs. 10/11/33	Orbit and scalp	No treatment here, elsewhere over an eight year period	Died 10/15/33	Undoubtedly there was too much reliance upon irradiation and potassium iodide.
(3) J. G.—622467 Male—65 yrs. 10/20/33	Entire right side face, neck and scalp, three years' duration	Roentgen ray, radium, and an occasional incision	Died 7/7/35	Died of an actinomycotic meningitis; a far advanced case when first seen. Exenteration of contents of right orbit may have obviated fatal meningitis. All facial actinomycosis except that of orbit under control at time of death. There was also found involvement of right middle ear at postmortem examination.
Recovered cases:				
(4) A. D.—38703 Male—65 yrs. 1/30/27	Right side of face and neck	Multiple incisions, drainage, KI and roentgen ray	Well	
(5) L. C.—42646 Male—34 years. 1/6/28	Right side of face and neck	Incisions, KI, and roentgen ray	Well	
(6) M. B.—567683 Male—43 yrs. 6/25/30	Tongue	Incisions, drainage, KI, and roentgen ray	Well	
(7) A. G.—55891 (Fig. 2) Male—40 yrs. 10/6/30	Large lesion in right cheek, unable to open mouth	One surgical excision	Well	Temporary parotid fistula, healed with small residual scar; function good.
(8) G. M.—62058 Male—46 yrs. 1/5/32	Right side of face, ear, and neck, three years' duration	One curettement; two excisions	Well	
(9) A. S.—60976 Female—29 yrs. 1/5/32	Large mass of recent origin beneath left jaw	Two excisions	Well	
(10) C. K.—611438 Male—39 yrs. 11/14/32	Tongue, of short duration	One incision and packing of tongue	Well	
(11) I. O.—608171 (Fig. 3) Female—13 yrs. 7/30/32	Entire right side of face, three months' duration	Two curettements, subsequent excision of scar	Well	
(12) M. W.—631475 Female—35 yrs. 9/27/34	Right side of face and neck	One excision, wound left open to heal by granulation	Well	
(13) A. B.—631495 (Fig. 4) Male—7 yrs. 9/28/34	Right cheek, right side neck; behind right ear	Two curettements	Well	
(14) L. H.—642452 Male—52 yrs. 9/29/35	Left cheek, left side neck from sternum to ear with recent	Two excisions of tissue including greater portion of	Well	Seven months' treatment elsewhere with intensive irradiation and KI and opening o abscesses. Family had

	progression to right side of neck	left sternocleidomastoid muscle and one curettement		been told that prognosis was very poor. Under treatment the patient had only become more apathetic. Weight loss 50 pounds. Four months after first evacuation of actinomycotic dead tissue here, wound was healed and patient had regained weight and vigor.

* In all but the doubtful cases (Table IV), *Actinomyces* were demonstrated morphologically and culturally (*Actinomyces bovis*). The writer here wishes to acknowledge the help which the keen interest of Dr. A. T. Henrici, Professor of Bacteriology, and his associates have afforded in the identification of these cases.

TABLE II. THORACIC CASES

(1) E. A.—49008 Male—21 yrs. 2/29/29	Left lung and chest wall	KI and roentgen rays	Died after leaving hospital	
(2) A. P.—55740 Male—13 yrs. 9/25/30	Suppurative pneumonitic pericarditis and mediastinitis with compression of the esophagus	Bed rest	Died 1/19/31	Actinomycotic nature of lesion not apparent until autopsy.
(3) E. B.—615300 (57754) Female—29 yrs. 2/14/31 (Fig. 5)	Left lung, chest wall and breast	Numerous rib resections and subsequent curettements	No evidence of residual actinomycosis during last 18 months of observation. Died of brain abscess	Brain abscess may have been actinomycotic; no autopsy. Patient did have serious pansinusitis before symptoms of cerebral abscess developed. (Case reported in detail elsewhere.)
(4) J. S.—612412 Male—15 yrs. 12/10/32	Left chest wall with supradiaphragmatic collection in both chests and involvement of spleen and liver	Bed rest, incision of abscesses over left chest wall and excision of one rib	Died 2/4/33	Presented as a case of actinomycosis of left thorax, but probably began as abdominal actinomycosis. Abscesses of liver were prominent at autopsy.
(5) L. W.—640072* Female—15 yrs. 7/2/35	Left lung, pleura, and chest wall, including left breasts, left axilla. Actinomycotic abscesses of left groin and left buttock	Incision, rib resections, curettements, packing and transfusions	Still under treatment	
(6) J. N.—646103 Male—57 yrs. 2/18/36	Right lung, suppurative pericarditis, and actinomycotic abscess of right kidney	Bed rest	Died 2/28/36	Presented as thoracic actinomycosis, probably an antecedent abdominal lesion
(7) J. L.—646743* Male—45 yrs. 3/10/36	Right lung, pleura, chest wall, and subdiaphragmatic collection	Excisions, curettements, packings, transfusions	Still under treatment	Antecedent appendectomy about seven months before, followed by empyema which was drained elsewhere. Free bleeding of sinus tract suggested diagnosis.

* See footnote on page 763.

TABLE III. ABDOMINAL CASES

Patient, Hospital Number, Sex, Age, Admission Date	Extent of Lesion	Treatment	Result	Remarks
(1) J. T.—12506 Male—37 yrs. 10/23/17	Fecal fistula of abdominal wall, abscess and retroperitoneal collection	Cautery, drainage, packing	Died 6/14/18	Antecedent appendectomy.
(2) H. N.—50181 Male—27 yrs. 10/4/29	Abdominal wall with peritonitis of pubis and induration of right thigh and buttock	Incisions, KI, and roentgen ray	Died	Operated upon for appendicitis one year before.
(3) A. M.—56820 Female—51 yrs. 12/7/30	Sinuses in abdominal wall with large retroperitoneal mass	Incisions, KI, and roentgen ray	Died	Appendectomy for acute suppurative appendicitis two and one-half years previously. At time of drainage here, peritoneal cavity was opened and drained, and fistula developed.
(4) W. J.— 613527 (Fig. 6) Male—59 yrs. 1/6/33	Right lower abdominal wall and retroperitoneal region extending from pelvis to diaphragm, including liver	Two curettements and packing	Still under treatment. Sinus still open; general condition satisfactory	Antecedent operations with drainage for acute suppurative appendicitis, followed by large incisional hernia (Fig. 6A).
(5) L. B.—640854 Male—28 yrs. 7/30/35	Abdominal wall and retroperitoneal region extending from pelvis to diaphragm, including liver	Excision of visible and accessory lesions, transfusion	Died 1/9/36	Six months prior to first admission, patient had appendectomy for suppurative appendicitis with abscess; developed fecal fistula.

(See also Thoracic Cases: Case 4, J. S.; Case 6, J. N.; and Case 7, J. L.)

TABLE IV. DOUBTFUL CASES*

(1) E. C.—64853 Male—16 yrs. 7/19/32	Tongue	Incised by suprahyoid approach, with excision of abscess and tract in tongue	Well	Painful swelling with pain over two and one-half years with previous operation. Chronic abscess of doubtful origin. *Actinomyces* not found.
(2) J. N.—611778 Male—46 yrs. 11/25/32	Tongue	Intra-oral incision into tongue and packing	Cured (Died two years after hospitalization in gasoline explosion)	Clinical diagnosis, actinomycosis. Sulphur granules present. No *Actinomyces* demonstrated. Final diagnosis, therefore, abscess of tongue, probably pyogenic in nature.
(3) M. L.—613369 Female—28 yrs. 2/6/33	Fecal fistula with dissection tract going down into pelvis	Curettement	Well	No *Actinomyces* found. Histologic diagnosis, chronic inflammation. Fistula developed spontaneously with subsequent abscess in abdominal wall. No antecedent intraperitoneal operations.
(4) M. L.—645356 (Fig. 7) Male—20 yrs. 1/20/36	Osteomyelitis of the right scapula with sinuses near spine and tip of scapula	Curettement	Well	Mycelia with peripheral clubs demonstrated which were identical with *Antinomyces*. On culture, only *Staphylococci* grew.

* Clinically, actinomycosis appeared to be the most likely diagnosis. These cases were classified as doubtful when morphologic and cultural studies failed to show the presence of *Actinomyces*.

abdominal localization of the disease are well known. A patient with thoracic actinomycosis, which I reported in 1932, has since died of what her physician interpreted to be a brain abscess (Table II, Case 3). Since she first came to the hospital in 1931, she returned at six month intervals for observation. During the last 18 months of life, no evidence of residual actinomycosis could be detected. Unfortunately, postmortem examination was not obtained, and whether the abscess was actinomycotic will never be known. A surgical procedure in 1932 on the lung, moreover, was followed by the signs of an intracranial abscess in the left motor (hand) area which cleared up completely; the propagation of an actinomycotic thrombus to the brain is well known. However, shortly before her fatal illness, her physician informed me, she had a very severe pansinusitis.

At present I have two patients under treatment for thoracic actinomycosis. One of these undoubtedly began as an abdominal actinomycosis; subphrenic infection with penetration of the diaphragm, the establishment of an empyema and perforation of the chest wall occurred (Table II, Case 7). There is no divining-rod which will tell whether all the foci have been uncovered; and the tendency for the disease to burrow in every direction is well known. The problem with these cases is early recognition of the presence of actinomycosis, evacuation of the exudate and dead tissue—including all the pockets. In those instances in which the diseased process remains well localized, and particularly if dissection towards the surface occurs, prompt and adequate treatment is likely to bring about a cure.

Another patient (Table II, Case 5) with extensive actinomycosis of the thorax is also still under treatment. This process involved the left lung and the greater portion of the left pleural cavity, as well as a considerable portion of the left chest wall. An actinomycotic abscess of the left axilla, another in the groin, and one over the left half of the sacrum have also been evacuated. The greater portion of the entire left chest is bare and the scapula as well as breast is separated from the chest wall. Despite the unusual extent of the lesion and its obviously discouraging features, I have not wholly despaired of being able to do something for this young girl.* Potassium iodide and irradiation, which leave in their wake anorexia and frequent vomiting, I have not looked upon as promising enough to warrant a trial. The contents of abscesses are curetted away as they appear; the wounds being extensive, the changing of dressings is a painful procedure. Every third day, under brief intravenous evipal anesthesia, the wounds are carefully reëxamined and dressed. Dakin's solution, dichloramine T, and zinc peroxide have proved most efficacious in keeping these wounds clean.

Important Problems Relating to Actinomycosis.—The most signifi-

*Since this was written both these patients have died. Case 5, Table II had a small residual process in the left lung. No evidence of actinomycosis was found in the other previous areas of involvement. Case 7, Table II also had a residual process in the lung, and, in addition, a small hepatic abscess (1×2 cm.) just beneath the surface of the dome of the liver.

FIG. 7.—(A) A patient with a spontaneous cecal fistula and abscess in the abdominal wall. This was not proved to be actinomycosis, but it is not unlikely that it was (Table IV, Case 3). (B) After injection of an opaque substance into the fistulous tract. Communication with the cecum is demonstrated. (C) After two curettements, the fistula closed.

cant issue demanding solution concerning actinomycosis is its etiology. As was previously mentioned, the general opinion is that actinomycosis is an exogenous infection. There are those who hold, however, that it is endogenous in origin, from organisms within the mouth. Naeslund[26, 27] would compose these conflicting opinions by findings of his own which suggest that actinomycosis may be caused by the organisms which inhabit the mouth as well as by the *Actinomyces bovis*. The former, he says, gives rise to much less serious infections. This is essentially a bacteriologic problem and one which I do not feel competent to discuss. Its solution is vital for the formulation of intelligent efforts directed at preventing the disease. When it is definitely known how the disease reaches man and gets its start, an important step will have been taken in the direction of prophylaxis of the disease.

Next in importance is early recognition of the presence of actinomycosis —particularly visceral manifestations of the disease. Almost invariably an extensive suppurative process is present in the thoracic and abdominal forms before the disease is identified. Efforts have been made to detect the presence of actinomycosis by cutaneous reactions similar in character to the Mantoux test for tuberculosis, but there is no evidence that such tests at present are worth while. When suppuration has already occurred, employment of the well known bacteriologic and pathologic criteria of examination should insure recognition of the diseased process. Early recognition and early evacuation of the dead tissue will prevent dissection and obviate the collection of exudate in pockets remote from one another. It is this occurrence which accounts for the poor prognosis in the visceral forms of the disease. By the time the nature of the suppurative process is properly identified, widespread dissection has occurred. And unlike a pyogenic abscess, tenderness is frequently absent following initial drainage. A residual pocket, even though in the proximity of the drainage tract, may not be uncovered until it later ruptures into the sinus of the drainage tract. Unfortunately, in the meanwhile, it is also extending itself in other directions. It is highly important, therefore, that actinomycotic wounds be frequently and meticulously examined. When a pocket has been well curetted, persistent drainage usually means an adjacent focus.

The manner in which the wound is treated is probably of far less importance than early drainage of all the pockets. Inasmuch as the organism is an anaerobe, zinc peroxide, as advised by Meleney[25] for anaerobic infections, should be useful. In the main, however, the consideration of most consequence is whether all the actinomycotic débris has been curetted away.

SUMMARY AND CONCLUSIONS

The cases of actinomycosis which have been seen at the University Hospital are reviewed. Examination of the data pertaining to them indicates that the prognosis of cervicofacial actinomycosis is good unless too much reliance is placed on the therapeutic response to the administration of potassium iodide and the employment of irradiation. The prognosis in thoracic and

abdominal actinomycosis is poor. With earlier recognition and adequate evacuation of dead tissue in visceral forms of the disease, the outlook will probably improve.

The most direct agency in the treatment of actinomycosis is surgery. The rationale of surgical treatment lies in the fact that the infection is essentially an anaerobic one. Removal of the dead tissue, which is poorly oxygenated and in consequence an excellent culture medium, will usually terminate the disease. Many instances of cervicofacial actinomycosis respond favorably to curettement alone. In extensive cases, energetic surgical excision of devitalized tissue is indicated.

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THE SURGICAL TREATMENT OF ESSENTIAL HYPERTENSION

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It is obviously impossible to present, in any comprehensive way, a discussion of the problems of essential hypertension and of its surgical treatment in such an abridged article as this. A fairly complete report of the work which Dr. Irving Page and I have done thus far has been prepared and will appear shortly, to which reference may be made by those who are interested. I wish to report, however, briefly the considerations which led to the adoption of certain surgical procedures, the selection of patients for operation, the methods of control used in studying patients before and after operation, and the results of operation which thus far have been obtained.

The time at which Doctor Page and I became associated in this work, early in 1934, was a period of great activity in the surgical treatment of hypertension; and a survey of the literature showed that American, French, German and Italian authors had published a variety of surgical and other procedures for its cure or amelioration. The therapeutic procedures described consisted in the reduction of the activity of the basophilic cells of the hypophysis by radiation, in denervation of the adrenal glands, in subtotal adrenalectomy, in splanchnicotomy and in anterior nerve root section. While the causation of essential hypertension was, and is not, known, the theories upon which these therapeutic procedures were based were chiefly three: that hypophyseal basophilism played an important rôle in the genesis of the disease; that overactivity of the adrenal glands resulting in a hyperadrenalemia was an essential factor; and that chronic spasm of the arterioles in the splanchnic area was an important cause of the elevated blood pressure. Evidence of whatever nature in support of these theories was sought and subjected to critical analysis; and as a result Page and I concluded that a surgical procedure which had as its purpose the relaxation of the presumably spastic arterioles of the splanchnic area and the denervation of the adrenal glands offered greater possibilities than others which had been suggested and tried. The choice of procedures, then, lay between splanchnicotomy and anterior nerve root section and we decided to make as accurate a study as possible of the results of both procedures. That we selected anterior nerve root section for our first study was the result of a survey of available anatomic and physiologic data. Even granting the possibilities of anatomic abnormalities and "overlap" in the distribution of the sympathetic nerves, it seemed to us that section of the sympathetic fibers at their point of emergence from the cord by division of the sixth dorsal to the second lumbar motor spinal nerves was a more certain method of inhibiting the sympathetic innervation of the

abdominal blood vessels and adrenal glands than section or resection of major and minor splanchnic nerves combined with lower dorsal ganglionectomy. The procedure was therefore attempted or carried out in 21 cases. At the time of the first operation, May 11, 1934, the only case of which we knew who had been treated by this method had just been reported. This was the case of Adson and Brown, and while the results were far from ideal, it was thought that the fault lay in the selection of the case and that the operation should be tried in cases in which the vascular tree was still flexible. It was realized that the operation was the most difficult, the most time consuming and the most dangerous of the procedures which had been suggested. That our fears of the possibilities of harm to patients in this operation have been to some extent realized, I shall indicate later.

In the selection of patients for operation, it seemed probable that those in whom an elevated blood pressure was the only objective manifestation of disease on physical examination, might be expected to respond most favorably to surgical treatment. Nevertheless, it was thought desirable to attempt the procedure in groups of cases, the groups representing stages in the severity of the disease; for aside from determining its value it seemed important to determine, if possible, its limitations. In determining groups of this sort, various criteria were used. The flexibility of the vascular tree was determined by the fall in blood pressure when the patient was put to bed; by the fall in blood pressure after the intramuscular injection of colloidal sulphur or acetyl-β-methylcholine (mecholine); after administration of sodium thiocyanate by mouth and after the inhalation of amyl nitrate; and by the daily fluctuations in the level of the blood pressure as determined by a long period of observation. Evidences of cardiac damage, of extensive changes in the ocular fundus and of renal damage with renal insufficiency were criteria which, in addition to vascular flexibility, were used to indicate the stage of the disease or its degree of advancement in the patients who were studied and subjected to operation. Such patients represented examples of the disease which varied from the benign essential hypertension of short duration to the highly malignant hypertension.

The prolonged observation and scientific study of patients before and after operation seemed most essential in determining the value of our therapeutic procedures. A study of all case reports in the literature showed the extraordinary difficulties in evaluating the results of different surgical operations, and largely because of the paucity of reported observations before and after operation. The great variability in the manifestations of essential hypertension makes it necessary that repeated observations, comprehensive in their scope, be made by one familiar with the disease in order to determine accurately the effects of any given procedure. What value there is in our work largely lies, I think, in the accuracy and completeness of the observations made before and after operation. The patients subjected to this study were first hospitalized in the Hospital of the Rockefeller Institute for at least 30 days, and frequently for a longer period, for a preliminary or preoperative

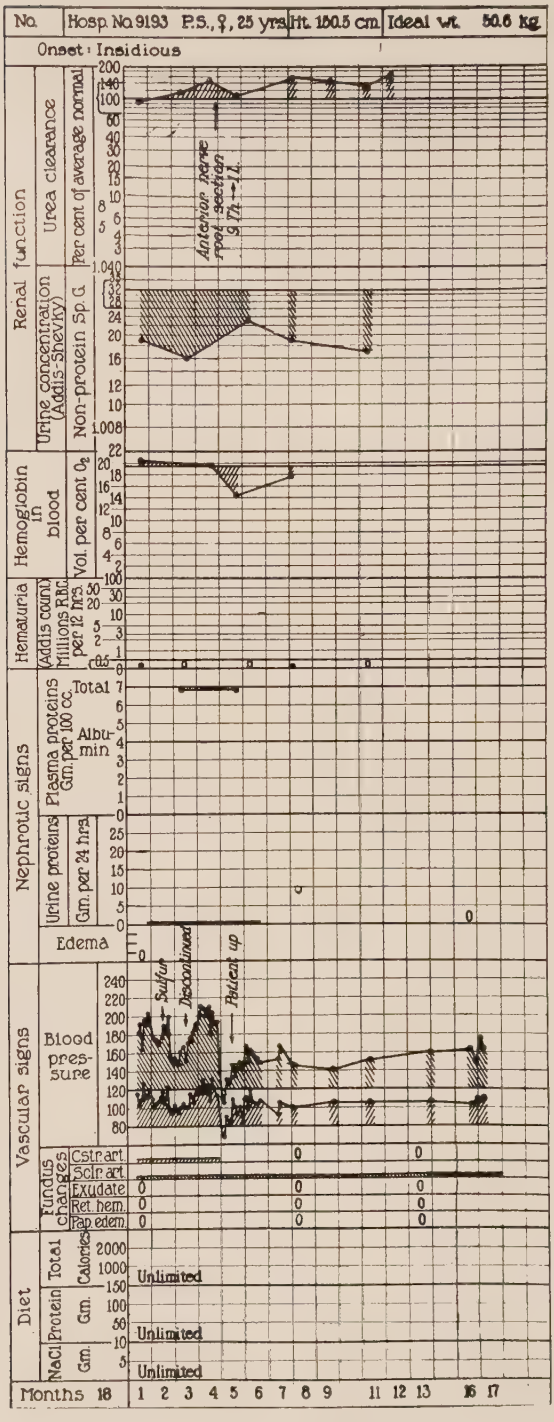

CHART 1.—Showing the observations made before and after the operation of anterior nerve root section. It will be noted that the blood pressure has remained at a lower level since operation.

period of study. The flexibility of the vascular tree was estimated by the tests above indicated. The blood pressure was taken daily at a fixed time (9:30 A.M.) with the patient in bed, its fluctuations established and a mean or average blood pressure determined. Changes in the eyegrounds noted were constriction of the arterioles, arteriosclerosis, exudates, hemorrhages and papilledema. Changes in the heart were determined by physical examination, measurements of roentgenograms, and electrocardiographic studies. The basal metabolic rate was regularly determined. Renal efficiency was measured by urea clearance and the ability of the kidneys to concentrate urine. The specific gravity of the urine was determined in a 12 hour specimen voided at the end of 24 hours without fluids; in case proteins were present in sufficient amounts to contribute to the specific gravity, a correction was made for it. The number of formed elements in the urine was determined by the technic of Addis. The urine protein was measured by the method of Shevky and Stafford as modified by MacKay. The plasma proteins were ascertained by the method of Howe; hemoglobin was measured by the method of Van Slyke and Neill. The general study of the patient included observations upon the subjective manifestations of the disease, upon psychic abnormalities and upon evidences of stability of the nervous system. After convalescence from operation, the patients were again transferred to the Hospital of the Rockefeller Institute and all the above observations and examinations repeated. In the follow up, the patients have periodically been hospitalized for a week or ten days and again studied. It would appear that the results we have to report are as accurate as can be determined in the human subject with known methods of examination. An example of the data record incorporating these observations is shown in Chart 1.

The operation of anterior nerve root section consists in the bilateral division of the anterior roots of the sixth dorsal to the second lumbar spinal nerves. The positive identification of the spinous process of the first lumbar vertebra and its relation to the overlying skin has been helpful in determining the lower limit of the incision. The identification of the last digitation of the dentate ligament after the dura has been opened has aided in determining the roots to be divided; for it is attached to the dura between the twelfth dorsal and the first lumbar nerves. In our early experience the entire operation was performed at one sitting. The disadvantages of this were found to be chiefly two: its duration and its consequent hardship upon both patient and surgeon; but more important the fall in blood pressure as the result of the laminectomy, so that the essential part of the operation—the division of the anterior nerve roots—sometimes could not completely be carried out. In our later experience the operation has been divided into two stages, the first terminating with the completion of the laminectomy and the complete exposure of the dura; the second consisting in the opening of the dura and the division of the anterior nerve roots. An interval of several days to a week is allowed to elapse between these stages, during which the blood pressure will have returned to its preoperative level. In the division of the

nerve roots every precaution has been used to avoid injury to the cord and every effort has been made to preserve the small nutrient blood vessels entering the cord along the nerve roots. A sketch of the essential part of the operation is shown in Figure 1.

FIG. 1.—The operation of anterior nerve root section. The dura mater is held apart with a series of silk sutures. The clamps grasp the digitations of the dentate ligament, traction upon which rotates the cord, and bring the anterior spinal roots into view. An anterior nerve root is shown divided between ligatures of fine silk. A blunt hook is shown lifting up a root preparatory to ligation and section.

The operation of splanchnic nerve resection has been performed according to the technic of Max Peet. It is a supradiaphragmatic approach through a vertical incision with the subperiosteal resection of the mesial portion of the eleventh rib. The pleura is stripped from the bodies of the vertebrae, the major splanchnic nerve is identified and a section 7 to 8 cm. long is removed. The tenth, eleventh and twelfth dorsal ganglia with the minor splanchnic nerve are then removed. The operation has been performed bilaterally and has been done either at one or at two sittings, separated by a week's interval. The subdiaphragmatic approach suggested and used by Craig and the more recent subdiaphragmatic approach with resection of the splanchnic nerves, two upper lumbar ganglia and portions of the adrenal glands, as described by Adson, have not yet been employed by us.

In the reports of results of various surgical procedures, observers have commented upon both subjective and objective improvement. By subjective improvement is meant the amelioration or disappearance of such symptoms as headache, vertigo and palpitation of the heart; by objective improvement, the actual lowering of blood pressure, disappearance of papilledema, and so forth. Experience shows that improvement in subjective symptoms may take place without marked lowering of the blood pressure; it shows also that improvement in subjective symptoms practically always occurs in conjunction *with* a fall in the blood pressure. In an appraisal of the value of surgical procedures in the treatment of hypertension, it would seem obvious that procedures which cause a per-

manent or long continued reduction in the level of the blood pressure are the procedures of choice.

Results of Anterior Nerve Root Section.—Up to April 1, 1936, anterior nerve root section had been attempted in 18 cases. The results are summarized in Table I. Certain comments upon these cases may be added by way of elaboration of the data shown.

In three cases the operation was not carried beyond the first stage. One patient (No. 17 in Table I) recovered promptly from the operation but thus far has refused to submit to the second stage. It will be observed that his

TABLE I

Summary of Clinical Data of 18 Patients on whom Root Section was Performed.

No.	Age	Duration of Hypertension	Retinal Changes	Urea Clearance	Av. Blood Pressure before Operation	Number of Pairs of Roots cut	Av. Blood Pressure after Operation	Subjective Improvement
	yrs.			Percent of normal	mm. Hg.		mm. Hg.	
1	23	18 mos.	0	104	190/122	9	150/94	Very marked
2	24	+ 2 yrs.	+	80	206/148	5	162/98	Very marked
3	17	18 mos.	+	106	180/122	5	140/90	Very marked
4	32	8 yrs.	0	101	210/130	5	154/104	Marked
5	35	3 yrs.	+	88	190/122	6	176/114	Marked
6	25	3 yrs.	+	91	184/116	9	156/106	Marked
7	40	2 yrs.	++	72	230/142	4	210/122	Marked
8	25	2 yrs.	++	74	210/130	6	162/110	Marked
9	24	7 mos.	++++	92	190/120	5	162/110	Marked
10	26	+ 3 mos.	++++	30	190/124	5	190/138	No symptoms before op.
11	33	10 yrs.	0	90	258/140	6	182/116	Moderate
12	46	15 yrs.	+++	68	270/160	5	230/142	Marked
13	37	2 yrs.	++++	55	270/170	6	210/140	Moderate
14	44	3 yrs.	++++	88	200/152	6	158/106	Marked
15	26	1 yr.	++++	18	200/110	7	Patient died shortly after dura was closed.	
16	21	2 yrs.	0	119	200/132	0	140/100	No symptoms before op.
17	37	7 yrs.	+	42	230/140	0	232/140	Questionable
18	39	6 yrs.	+++	86	270/148	0	Patient died from Streptococcus Meningitis	

* The elapsed time since operation in the first 14 cases varies between 1 and 2 years.

blood pressure remains at the preoperative level. Clinically he represented an advanced stage of the disease. One patient (No. 18) developed symptoms of meningeal irritation following the first stage and died from a Streptococcus meningitis. The dura had been opened only in the sense that a hypodermic needle had penetrated it for the purpose of withdrawing some cerebrospinal fluid. The meningitis probably was the result of a gross error in technic. One patient (No. 16), during his recovery from general anesthesia after the first stage, is said by two observers to have moved his legs; but when observed the morning following operation there was clear evidence of a transverse lesion of the cord at the level of the upper end of the incision. Believing that extradural hemorrhage causing compression of the cord might be the cause of his condition, the wound was reopened and a considerable clot was evacuated. There was no hemorrhage beneath the dura, as demonstrated by its color and by the aspiration of clear cerebro-

spinal fluid. Hoping that the extradural clot was sufficient to explain the symptoms of cord compression, the wound was again closed without opening the dura. For a period of eight days we awaited some evidence of returning function; then reopened the wound, removed the laminae of two dorsal vertebrae above the upper limit of our previous exposure and opened the dura throughout the entire length of the operative defect in the spine. There was not the slightest hemorrhage beneath the dura and the most careful examination of the spinal cord failed to reveal any trauma of the external surface of the cord or evidence of hemorrhage or other lesions within the cord. Since this operation (three months) there has been a slow return of function; but that there will be a restoration to normal function seems at present very doubtful. It is to be noted that as the result of a cord lesion at the level of the sixth dorsal, a fall in blood pressure has occurred and persisted as in cases in which the anterior nerve roots have been divided.

In 15 cases the operation of anterior nerve root section was completed in one or two stages. One patient died at the termination of an operation performed in one stage; and for want of a better explanation the death is ascribed to surgical shock. The autopsy showed generalized arteriosclerosis, arteriolar nephrosclerosis, detachment of the retinae with retinal hemorrhage, partial atelectasis of the lungs, persistent thymus and cholelithiasis. The case was one of our early ones, and was the most advanced case of malignant hypertension in the series; it is doubtful, in retrospect, whether the operation should have been undertaken.

Of the 14 patients who survived the completed operation, three are improved, but the duration of time is not sufficient to warrant comment on the results; 14 have been observed for from one to two years since operation. Of these 14, five have roughly been grouped on the basis of duration of the disease, retinal changes, evidences of arteriosclerosis and of renal damage, as mild or moderately severe examples of essential hypertension; while nine have been classified as examples of advanced and severe hypertension or as malignant hypertension.

In Table I the first five cases represent the mild and moderately severe examples of the disease, the sixth to the fourteenth cases, inclusive, represent the advanced and severe examples of the disease. Subjective improvement has been very marked in three, marked in eight, and moderate in two of the cases. Improvement in subjective symptoms has, therefore, occurred in 100 per cent. Objective improvement—the lowering of the blood pressure during the period of observation, the improvement in the condition of the eye-grounds, and so forth—has varied appreciably, and in order to form a just estimate of the results, it is necessary to analyze each case separately. Perhaps a general idea of the results may be obtained from the appended summary.

Summary.—In four out of the five cases of mild or moderately severe hypertension, the systolic pressure before operation varied between 180 and 220 and averaged 196.5; the diastolic pressure before operation varied be-

tween 122 and 148 and averaged 130.5. The permanent (during period of observation) fall in systolic pressure after operation varied between 40 and 56 Mm. and averages 45 Mm. The permanent fall in diastolic pressure after operation varied between 26 and 50 Mm. and averages 34 Mm. The lowering of the blood pressure represents 22.6 per cent of the preoperative systolic level and 25.5 per cent of the preoperative diastolic level. The patients represent a group which, having had a blood pressure before operation of 196/130, have had since operation a blood pressure of 150/95; a blood pressure which approaches, but is not, normal.

In one of the cases of mild or moderately severe hypertension, the blood pressure before operation was 190/122 and since operation has been 176/114. There has been, therefore, a fall of only 14 Mm. in the systolic pressure and 8 Mm. in the diastolic pressure. The result in this case is far less satisfactory than in the other four.

In four of the nine cases of advanced, severe hypertension, the systolic pressure before operation varied between 200 and 270 and averaged 235; the diastolic pressure before operation varied between 130 and 170 and averaged 143. The permanent fall in systolic pressure after operation varied between 42 and 76 and averaged 56.5 Mm.; the permanent fall in diastolic pressure after operation varied between 20 and 30 and averaged 25 Mm. This lowering of blood pressure represents 23.8 per cent of the preoperative systolic level and 17.5 per cent of the preoperative diastolic level. The patients represent a group which, having had before operation a blood pressure of 235/145, have had since operation a blood pressure of 178/118, a pressure considerably above normal. It will be noted that the percentage fall in the systolic pressure in this group is equal to that in the mild and moderately severe group, but that the percentage fall in diastolic pressure is less than that in the previous group. Although the operation achieved the same percentage fall in systolic pressure as in the preceding more favorable group, the fact that the blood pressure before operation was considerably more elevated leaves this group with a greater degree of hypertension. (One of these patients has since died of apoplexy. That the reduction in blood pressure prolonged his life cannot of course be stated; that the operation hastened his end we do not think likely.)

In four of the nine cases of advanced, severe hypertension, the systolic pressure before operation varied between 184 and 270 and averaged 218; the diastolic pressure before operation varied between 116 and 160 and averaged 134.5. The permanent fall in systolic pressure after operation varied between 20 and 40 and averages 29 Mm.; the permanent fall in diastolic pressure after operation varied between 10 and 20 and averages 14.5 Mm. This lowering of blood pressure represents 13.3 per cent of the preoperative systolic level and 10.5 per cent of the preoperative diastolic level. The patients represent a group which, having had before operation a blood pressure of 218/135, have had since operation a blood pressure of 189/120, a pressure again considerably above normal. It is to be noted in

this group that the percentage fall in systolic and diastolic pressures following operation is very much less than in the preceding two groups of cases. (Of these cases one has since died of apoplexy.)

In one of the nine cases of advanced, severe hypertension, the systolic pressure before operation was 190 and the diastolic pressure 124. Following operation the systolic pressure has continued to be 190 but the diastolic has increased to 138. The operation in this case has had no effect upon the blood pressure. (The patient has since died of uremia.)

It will be noted that in all cases except one, anterior nerve root section has been followed by a fall in blood pressure which has persisted for from one to two years. In mild and moderately severe hypertension, four out of five cases, or 80 per cent, have shown a satisfactory reduction in blood pressure. In severe and malignant hypertension, four out of nine cases, or approximately 45 per cent, have shown a fairly satisfactory reduction in blood pressure; while one out of nine, or ten per cent, has failed to show any reduction in blood pressure. The attempt to determine the reasons for fairly good results in 80 per cent of the mild and moderately severe cases and 45 per cent of the severe, advanced cases does not at present lead to any definite conclusions. Our experience thus far shows that neither in the mild nor severe cases can anterior root section be expected to cause a permanent reduction of more than 25 per cent of the preëxisting systolic and diastolic pressures. Perhaps in part because of this, we have found that patients whose disease is benign without advanced morbid vascular changes and young patients exhibiting signs of the hypertensive diencephalic syndrome are fairly certain to be greatly benefited by operation; patients with benign hypertension of long standing with marked arterial thickening form a more uncertain group from the viewpoint of results; while patients with malignant hypertension form a very uncertain group in whom the outcome cannot at present accurately be predicted.

Aside from the results with respect to the blood pressure, the objective findings following operation may be stated as follows: Morbid changes in the eyegrounds when present before operation quite generally disappeared. Relaxation of constricted retinal vessels, absorption of exudates and hemorrhages and reduction in the grade, or disappearance of papilledema was noted, not in all, but in many instances. The heart, as measured in preoperative and postoperative roentgenograms decreased in size after operation in a number of cases in which it was enlarged previous to operation. The renal function present before operation was not changed as a result of the operation.

The supposed untoward effects of the operation which might give rise to disabilities after operation have, thus far, in our experience been of very little significance. The paralysis of the abdominal muscles, the result of the section of the motor roots, was expected to give rise to a protuberant abdomen. While enlargement of the abdomen has occurred, it has been neither unsightly nor disabling. On the other hand, it has been observed that the blood pressure after operation in some cases is higher in the prone than in

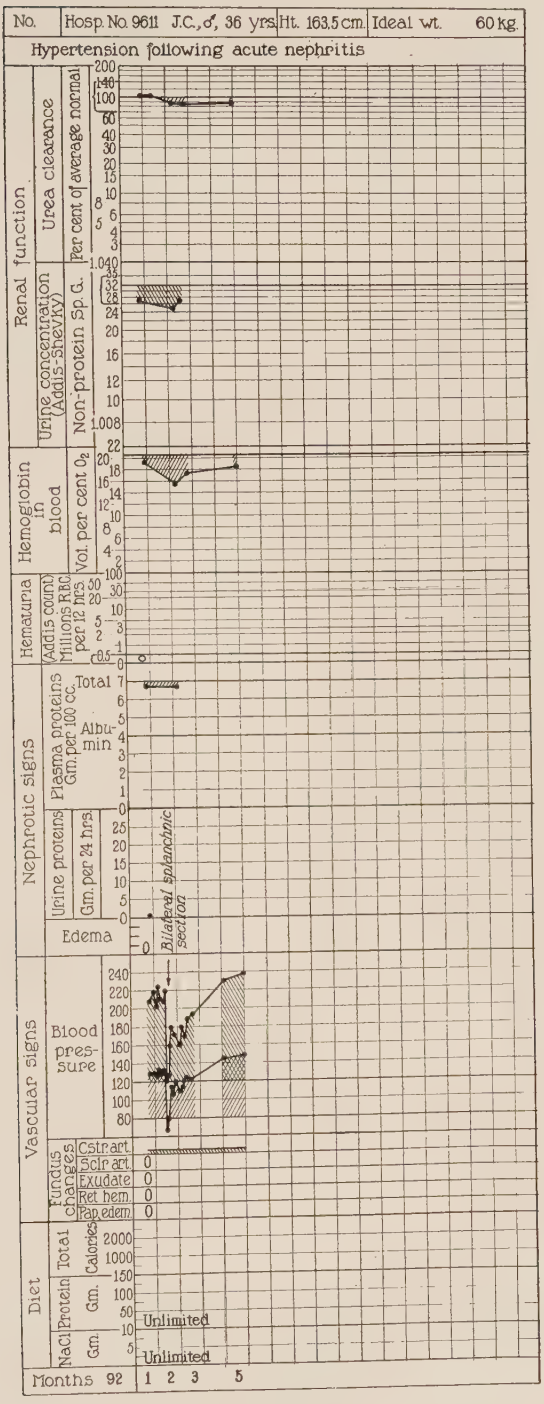

CHART 2.—Effects of splanchnic nerve resection. The blood pressure, reduced immediately after operation, has returned to its preoperative level. This has been our experience in nine cases subjected to this operation.

the erect posture, and it may be that relaxation of the abdomen is a factor in the lowering of the blood pressure. The loss of sweating of the skin of the trunk from the xiphoid or costal margin to the symphsis pubis has not been noticed by patients. The difficulty in evacuating bladder and bowel due to the loss of function of the abdominal muscles has disappeared, as a rule, within 48 or 72 hours. The supposed danger of failure of kidney function due to the fall in blood pressure was not anticipated by us and has not been realized. The blood flow through the kidney is maintained satisfactorily with a lowered blood pressure, and Doctor Page's postoperative studies show that the urea clearance and the ability to concentrate urine are practically unaffected by operation.

The serious danger in the operation is the occurrence of a lesion of the cord giving rise to paresis or paralysis of the lower extremities, bladder and rectum. The nature of the lesion in our case and its cause remain for the moment undetermined. That the lesion could have been due to cord compression the result of a blood clot in the wound seems doubtful; that it was not due to operative trauma of the cord seems proven by our careful examination of the cord; that it was not due to operative interference with the blood supply of the cord also seems clear. The possible occurrence of this complication seems to us a very strong objection to the procedure.

Our experience with the operation of splanchnic nerve resection combined with removal of the lower thoracic sympathetic ganglia can be stated very briefly. The operation in one or two stages has been performed thus far on nine patients and in all, except one instance, was carried out on both sides. In the selection of patients for this operation, seven of the nine cases had mild benign hypertension and two had malignant hypertension; the majority, therefore, were those in whom a favorable result might be anticipated. The same prolonged, careful and comprehensive observations have been made before and after operation as in the patients subjected to anterior root section. The results in the nine cases have been disappointing. In all, the blood pressure, reduced as a result of the operation, promptly rose to its preoperative level and for periods of six months to one year either has continued at this level or has become more elevated (Chart 2). The results from the viewpoint of the relief of subjective symptoms also have been minimal. So far as our experience goes, therefore, this procedure has failed to give results comparable with those of anterior root section; it is, however, realized that the number of cases thus far subjected to this procedure is too small from which to draw definite conclusions.

Discussion.—Dr. George W. Crile (Cleveland, Ohio).—This paper is distinctly a Heuer type of paper, careful and logical. For my part may I present a slightly different approach.

Let us take a view of the background, the possible source or genesis of hypertension. Figure 1 shows the adrenal gland and the sympathetic system in an alligator of about the same weight as a full grown lion or tiger. It

has no sympathetic complex whatever. Sympathin is a product of the sympathetic nervous system, which was discovered by T. R. Elliott, and has been found to function like adrenalin; that is, it instantaneously speeds oxidation, its effects spreading over the whole sympathetic system. If that be true,

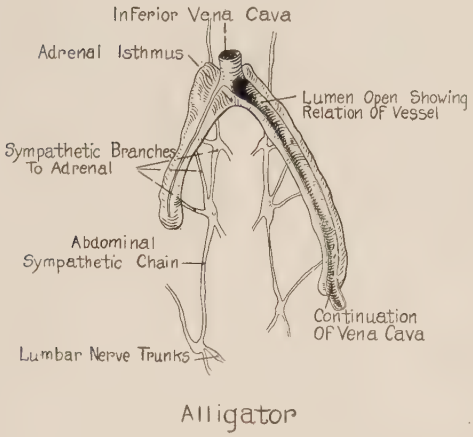

FIG. 1.—Adrenal sympathetic system of alligator.

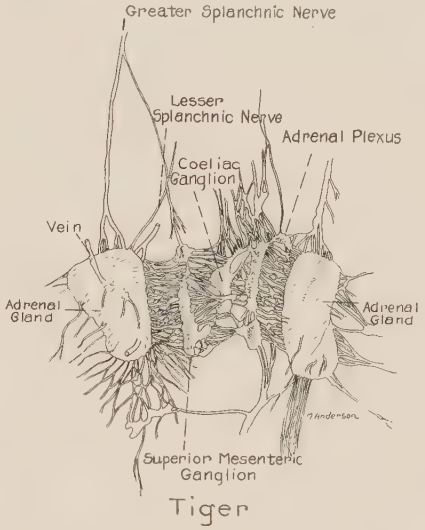

FIG. 2.—Adrenal sympathetic system of tiger.

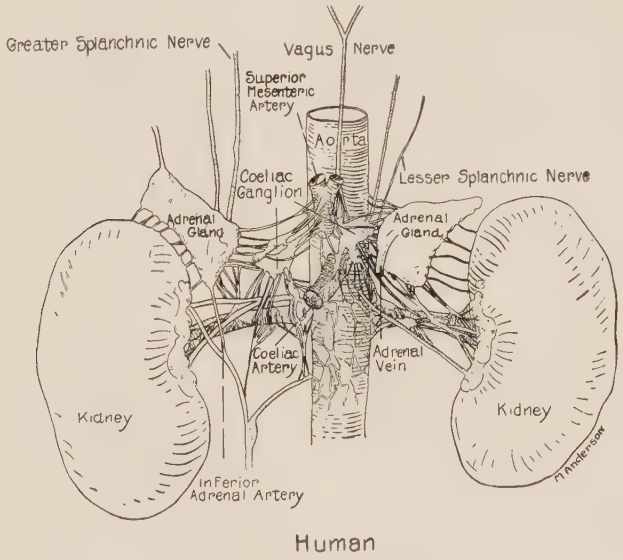

FIG. 3.—Human sympathetic system.

then those animals requiring great outbursts of activity should, in contradistinction to this slow and lazy alligator, have a great complex here. This contrast is exhibited in Figure 2, which shows the adrenal sympathetic system in a tiger of approximately the same weight as that of the alligator whose lack of a sympathetic complex we have just shown. Look at the enormous

development of the sympathetic complex, the numbers of unnamed ganglia present, like a cluster of grapes, and the size of the complex itself. Eighty-one fibers enter the adrenal gland in this powerful animal which shows the greatest power in its immediate outbursts of energy.

Figure 3 shows the human adrenal sympathetic complex. When we denervated the adrenal glands, we could cure hyperthyroidism and neurocirculatory asthenia. We could abate essential hypertension in some cases but in many cases we failed to do so and so we were dissatisfied. As the results of our clinical experience and our findings in animals during the last six months, we have concluded that there are at least two separate functions of this great energy system. One is that of driving—speeding the oxidation in the animal. That is clear enough. The other is a drive on the circulation,

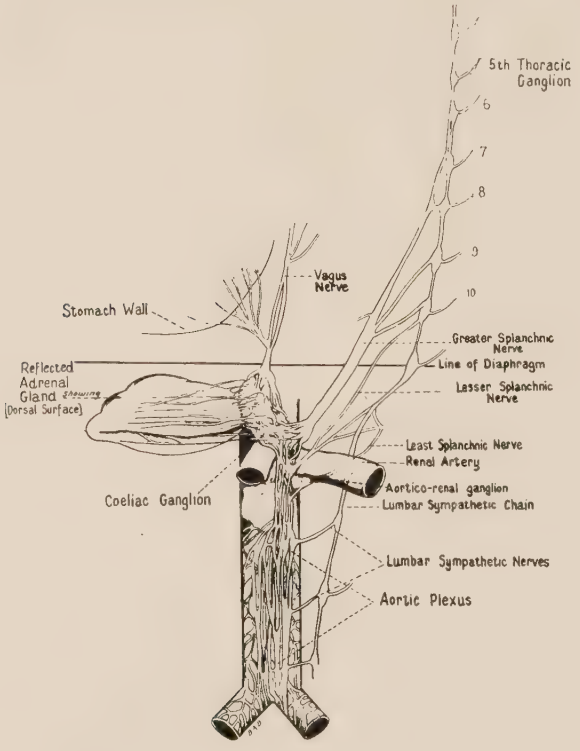

FIG. 4.—Sympathetic nerves on surface of aorta.

to carry an increased supply of oxygen through the blood stream. And to accomplish this we have a concentration of lines of communication on the aorta, some actually entering into its walls. There are similar communications with the whole arterial tree, the capillaries alone, according to Crowe, amounting in length to about 150 miles. We therefore changed our point of view, and in the treatment of hypertension attacked only the lines of communication of this energy system with the arterial tree.

Figure 4 shows the denervation of the aorta, from the bifurcation to the crus of the diaphragm. We also break up the celiac plexus and take out the celiac ganglia. This is a complete denervation, so far as the artery is concerned and that is all we need to think about in essential hypertension. One can see how inadequate it is just to denervate the adrenals and divide the

splanchnic nerves. Our attack should be solely upon the energy that causes the contraction of this great arterial tree.

A universal Raynaud's disease is probably what essential hypertension is. If that is true, then this procedure should produce an immediate effect, an immediate fall of the blood pressure. As the result of this operation, there is a more definite immediate fall in the blood pressure, especially in the diastolic pressure, and the hypertension is more definitely relieved. For the final end-results we must await the confirmation of time.

Dr. Alfred Adson (Rochester, Minn.).—I do not think there is a great deal to add after Doctor Heuer has given his thorough discussion and Doctor Crile his contribution to the subject. However, my interest in the subject has been carried along for a number of years; perhaps I might add one or two points relative to the selection of patients for sympathectomy and relative to the choice of operation.

I wish that it were possible always to prognosticate definitely, preoperatively, which patients are suitable for, and which are going to respond to, extensive sympathectomy. All of us who have been carrying out these operations, I am sure, have had this experience: we have advised operation and have performed it upon a patient whom we though should respond favorably, only to be disappointed and see the pressure rise and the symptoms return at a later date. Equally true, we have accepted a patient for operation rather reluctantly because of marked retinitis and changes resulting from high pressure and have found, to our surprise, that the patient made remarkable improvement following operation.

I want to emphasize what Doctor Heuer called attention to, and that is the preoperative studies, because it is very evident that if there has been irreparable damage to the heart, kidneys, retinae and cerebral vessels, little is to be expected from operative interference.

I had observed early in the series that whenever a patient's blood pressure dropped, as Doctor Crile illustrated on his chart, following thorough anesthetization before operation, that patient was the one who responded most favorably and received the best results. Doctor Allen, who has taken Dr. George Brown's place in the section on vascular disease, took the cue and has carried out a number of studies, such as Doctor Heuer has referred to, with reference to the drop in pressure during rest and under the influence of barbiturates; but Doctor Allen carried it one step farther. He had these patients anesthetized with a barbiturate, such as pentothal sodium, or an amytal, and frequently found that the systolic blood pressure would drop to 130 or 120 Mm. of mercury and even as low as 100. During the ten minutes of anesthesia the diastolic pressures also fell to less than 100 Mm. of mercury.

In comparing the preoperative and postoperative changes, Doctor Allen found that preoperative drops in pressure corresponded to the postoperative pressures. It appears that the anesthesia test is one that may help in selecting suitable cases. Even though there are no evidences of irreparable damage in the kidneys or heart, there may be present a fixed change in the arterial wall which will not allow vasodilatation. Therefore negative renal function and blood tests are not necessarily assurances that the patient will respond to extensive sympathectomy.

The object of the operation, as Doctor Heuer has told us, has been to denervate a large vascular area, below the diaphragm, and to thoroughly denervate the suprarenal gland high, at the source of its innervation. Perhaps the most radical and most effective operation is extensive rhizotomy, because then the sympathetic fibers are divided as they leave the spinal cord. The

operation Doctor Peet has been performing is a much simpler and less extensive procedure. It does not include the rami to the upper two lumbar ganglia and only occasionally the rami from the twelfth dorsal root.

We have had two cases such as Doctor Heuer referred to; that is, transverse myelitis which developed following extensive rhizotomy. This led to the development of the subdiaphragmatic procedure, since the supradiaphragmatic operation did not include as many sympathetic fibers as I wanted to divide.

In developing the subdiaphragmatic procedure, it occurred to me that if it were possible to resect all three splanchnic nerves, major, minor and lesser, on both sides, and if it were possible to take out the first and second lumbar ganglia, I would then interrupt all of the sympathetic fibers carrying vasoconstrictive impulses to vessels below the diaphragm, just as is accomplished in rhizotomy. Therefore, I have discontinued rhizotomy and have employed the subdiaphragmatic operation. To date, we have carried out the procedure on 25 patients, operating first on one side, and ten days later on the opposite side.

In view of the experiences that other men have had with the suprarenal gland, and in the light of Doctor Crile's work on denervation, I included biopsy of the suprarenal gland in the first case, but in the others I included half of each suprarenal gland. I am not sure that this is necessary.

In our group of cases in which rhizotomy has been performed, I should say the results are poorer than those that Doctor Heuer has reported. This may be attributable to the fact that in our early experience we had no guide for accepting cases except trial and error. The group of cases in which rhizotomy was performed numbered 27, with two deaths. One patient died from a suprarenal tumor; one from meningitis. Thirteen patients obtained satisfactory results. In the series in which the subdiaphragmatic operation was performed, there were 25 cases with no postoperative deaths, nor has there been a death since operation. The first patient was operated upon in February, 1935. Three patients in this group have not responded well, since the pressures have returned to preoperative levels, while the pressures of others have dropped so low that it has been necessary to apply an abdominal binder.

I believe the subdiaphragmatic operation is just as effective in interrupting vasoconstrictor impulses as is rhizotomy, and it is possible that the operation may be more effective in that we are interrupting postganglionic rami instead of preganglionic fibers.

Dr. Dallas B. Phemister (Chicago, Ill.).—In general, the excision of normal structures, with obliteration of normal physiologic processes, should not cure disease as does excision of pathologic tissue obliterating a pathologico-physiologic process. We do not know but that the nerves excised or divided in these operations may be normal, and that is what makes it so difficult to determine what the effect is going to be upon the pathologic physiology.

I thought you might be interested in some experiments that Dr. Kieth Grimson has carried out with me, in which normal dogs were operated upon: excising the entire thoracolumbar chain on both sides, and then studying the blood pressure at frequent intervals by the arterial puncture technic, over long periods of time. After the first operation, *i.e.*, excision of the thoracic chain on the right side, a fall in blood pressure occurred; excision of the left side was then undertaken, followed by excision of the abdominal portion at one operation. The blood pressure remained low for a variable number of

days, ranging from 75 to 150; climbed gradually, and in 150 to 250 days, as a rule, was back to normal. In 430 days the pressure was practically the same as it was before the operation.

Autopsies have been performed upon some of these dogs, and there have been no evidences of restoration of the sympathetic chain. It is difficult to know whether there are fibers that have grown in and reestablished connections that cannot be identified grossly.

The same operation that Doctor Heuer has performed upon a patient, and also the Peet operation, was carried out upon dogs. In those cases, in both instances, there was a very rapid return of the blood pressure to normal. Four other dogs, operated upon by the method of Doctor Heuer, had a return of their blood pressure level in from 10 to 30 days.

These findings might be regarded as a discordant note in the surgical treatment of essential hypertension, but again I want to emphasize that the results obtained from surgery upon a normal dog are not comparable with results obtained, from the same operation, upon a sick patient.

THE DEMONSTRATION OF HORMONES IN TUMORS*

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The isolation of the pituitary and ovarian hormones has opened up new fields of experimental investigation as to the causes and processes of normal and pathologic growth. The significance of some of the results of these investigations is not yet understood. Herewith is recorded the results of bio-assays of the tissue of a number of different tumors. It was thought, in the beginning of this investigation of the hormones, that if the tissue changes associated with cystic disease of the breast and benign tumors were due to the ovarian hormones, these might, or probably could, be recovered from the tissue affected on bio-assay.

The effects of hormonal stimulation in normal development are generalized. A group of organs responds in a correlated manner to a given level of secretion and in a single organ all of the tissues composing it tend to respond to the same degree. This is quite different from tumor growth in which a single component in a localized focus of tissue exceeds in growth and amount the surrounding tissue in the remainder of the organ. However, it has been demonstrated by means of bio-assay that such a focus of actively growing cells may respond selectively and excessively to a biochemical stimulus of a hormone which usually is uniformly distributed in the blood.

Lewis and Geschickter[24] reported a localized concentration of the ovarian hormone, estrin, in a benign breast tumor (fibroadenoma) removed at operation, and showed that injections of this estrogenic hormone into monkeys produced hypertrophy of the breast. Extending this method of biochemical study to other human tumors, it has been shown that hormones secreted by the pituitary gland and the ovaries may be recovered in high concentrations from a variety of benign and malignant tumors. The method of assay consists of grinding up the freshly excised tumor and extracting the finely divided tissues with suitable solvents. The substances recovered by extraction are then injected into rats, mice or rabbits and the presence and amount of the hormones present determined by the effects on the sexual organs of the animals used for testing. These studies reveal that when high or normal levels of endocrine secretion are present in the blood or urine of patients, the tumor may contain excessive amounts of hormones probably concentrated locally by the growing tissues.

The presence in growing tissues of high concentrations of hormones has

* Aided by a grant from the Anna Fuller Fund.

but recently been demonstrated. Such concentrations had been found only in the tumors of organs which are normally endocrine in nature (organs which elaborate the hormone locally) such as the testicle, ovary or placenta. In the present studies such concentrations have been found in tumors of non-endocrine organs such as the breast, uterus, and bone.

Hormone Studies of Breast Tumors.—Castration was suggested as a treatment of carcinoma of the breast in women before the menopause by Beatson,[4] in 1896. He thought that castration caused fatty degeneration and death of the cancer cell. A few striking results were obtained in cancer by castration, but because of the number of failures and the higher percentage of cancer of the breast occurring after the menopause, this method of treatment was abandoned. In 1919, Loeb[26] came to the conclusion that the incidence of spontaneous cancer of the breast in inbred strains of mice could be reduced and ultimately prevented, if castration was carried out at progressively early stages in female mice. Loeb raised the question as to whether the relation of hormones to the development of cancer is specific, and whether a hormone influences the development of cancer only in those organs to which, under normal conditions, it has a definite relation. Cori,[10] in 1927, and Murray,[28] in 1928, supplied experimental evidence demonstrating that the ovaries and their endocrine secretions were of primary importance in raising breasts to the physiologic threshold where susceptibility to mammary carcinoma occurred in strains of mice susceptible to cancer of this organ.

Fig. 1.—Photograph of a girl, aged five, with enlargement of the breast and nipple following the injection of 6,000 rat units of estrin. The circles mark the limit of mammary enlargement.

A specific relationship between ovarian hormones and cancer of the breast, however, was not demonstrated until after the isolation of the ovarian hormone, folliculin or estrin, by Allen and Doisy[1] and other investigators. Lacassagne,[22] in 1932, demonstrated that injections of estrin caused carcinoma of the breast in male mice which otherwise remained cancer free, although the females of the same strain were susceptible to cancer of the breast. Lacassagne,[21, 23] in 1934, also found estrin in colostrum secreted from the breast of a woman with mammary carcinoma.

Virginal Hypertrophy, Gynecomastia and Fibro-Adenoma.—Enlargement of the breast associated with granulosa cell tumors of the ovary and high levels of estrin secretion have been repeatedly reported in the literature. Fig. 1 illustrates the action of estrin on the breast of a girl, aged five, who had received injections amounting to 6,000 rat units of estrin over a period of six

weeks in the treatment of gonorrheal vaginitis (Fig. 1). Apparently the tremendous hypertrophy of the breast observed at puberty (virginal hypertrophy) in certain women is due to increased secretion of estrin. In the one case in which we have had an opportunity to make an assay of the blood 25 rat units of estrin per liter were recovered.

Gynecomastia occurs in 5 per cent of the cases of teratoma testis. Lilienthal[25] has reported a striking case associated with chorio-epithelioma arising apparently in a mediastinal teratoma and accompanied by a positive Aschheim-Zondek test. In addition to the high concentration of the prolan found in the urine, both Heidrich and Hamberger have reported high concentrations of estrin in men suffering from gynecomastia and testicular tumors. We have demonstrated experimentally that mammary hypertrophy can be produced in male monkeys either through the direct action of estrin or indirectly by injections of prolan, the testicles being present. Moreover, in a case of gynecomastia occurring in a man of 22, we have demonstrated increased concentrations of estrin in the blood (10 rat units per liter) in the absence of any testicular neoplasm. In two other cases of gynecomastia, uncomplicated by testicular tumors, 200 and 2,500 rat units of estrin per kilogram have been recovered from the excised breast tissue.

TABLE I

BIO-ASSAYS FOR ESTRIN AND GONADOTROPIC SUBSTANCE IN FIBRO-ADENOMA, GYNECOMASTIA, INTRACANALICULAR MYXOMA, AND FIBROSARCOMA OF BREAST*

Subject	Estrin (Rat Units per Kilo)	Gonadotropic Substance (Rat Units per Kilo)
Rat: Control breasts	Negative	†
Rat: Fibro-adenoma	2,000	—
Rat: Fibrosarcoma	1,000	—
C. F. 19 Control breast	Negative	—
Menopause Control breast	Negative	—
B. Fibromyxoma	Negative	60,000
C. Fibromyxoma	200	—
C. Forro-adenoma	1,000	—
L. Fibro-adenoma	200	2,500
J. Fibro-adenoma	6,000	—
D. Fibro-adenoma	12,000	—
Co. Fibro-adenoma	2,000	—
Ba. Fibro-adenoma	18,000	—
Ku. Fibro-adenoma	250	—
M. Gynecomastia	200	Negative
K. Gynecomastia	2,500	Negative

* Tissues fixed in 95 per cent alcohol.

† Not done.

The localized tumors of the breast, such as fibro-adenomata, have a microscopic structure similar to that seen in diffuse virginal hypertrophy. In the belief that concentrations of estrin would be physiologically significant for the tumor growths we have assayed such tumors removed at operation. These assays show that the concentration of estrin may be as high as 18,000 rat units per kilogram. Giant fibromyxomata of the breast have also been assayed. These assays not only showed high concentrations of estrin but also as many as 60,000 rat units of gonadotropic substance per kilo (Tables I and II).

TABLE II

ASSAYS OF BLOOD AND URINE IN VIRGINAL HYPERTROPHY AND GYNECOMASTIA

Diagnosis	Estrin Rat Units per Liter	Gonadotropic Substance Rat Units per Liter
Gynecomastia (S)	Blood—Negative	Urine—Negative
Gynecomastia (K)	Blood—Negative	Urine—Negative
Gynecomastia (L)	Blood—25	Urine—Negative
Gynecomastia (W)*	Blood—Positive	Urine—40,000
Infantile hypertrophy (H)	Blood—Not done	Urine—Positive
Virginal hypertrophy (LKY)	Blood—25	Urine—Not done

*Associated with Teratoma Testic

Cystic Disease of the Breast.—In cystic disease of the breast there is an increase in the fibrous elements, and hyperplasia of the duct epithelium with dilatation of the ducts and cyst formation. Experimentally—cystic changes may be produced in the breast by the injection of estrin—and changes simulating lactation by the use of prolactin (Geschickter and Lewis).[14] It would seem that in cystic disease the combined effects of estrin and prolactin stimulation were represented. The assays of the tissue and cyst fluid, from the breast of patients with cystic disease, have revealed high concentrations of both estrin and prolactin (the lactogenic substance of the anterior pituitary) (Table III).

TABLE III

ASSAYS OF TISSUE AND FLUID FROM PATIENTS WITH CYSTIC DISEASE OF THE BREAST FOR LACTOGENIC AND ESTROGENIC SUBSTANCES

Specimen	Patient	Hormone Assayed	Results of Bio-Assay Bird Units per Liter
Cyst fluid*	S. K.	Lactogenic substance	2,000
	L. F.	Lactogenic substance	720
	Bur.	Lactogenic substance	0
	Jar.	Lactogenic substance	250
	Car.	Lactogenic substance	100
	Gar.	Lactogenic substance	750
	N. W.	Lactogenic substance	250
	Grom.	Lactogenic substance	250
	Wei.	Lactogenic substance	400

TABLE III—*Continued*

Specimen	Patient	Hormone Assayed	Results of Bio-Assay Bird Units per Liter
			Rat Units per Liter
Cyst fluid†	B. C.‡.....	Estrogenic substance	6,000
	Jar.....	Estrogenic substance	0
	Car.....	Estrogenic substance	2,000
	Gar.....	Estrogenic substance	0
	N. W.....	Estrogenic substance	0
	Joh.....	Estrogenic substance	1,000
			Rat Units per Kilogram
Mammary Tissue	N. B.....	Estrogenic substance	6,000
	R. C.....	Estrogenic substance	1,000
	P. D.....	Estrogenic substance	200
	I. D.....	Estrogenic substance	4,000
	Bro.....	Estrogenic substance	0
	A. L.....	Estrogenic substance	0
	Bur.....	Estrogenic substance	0
(Adenosis)	D.....	Estrogenic substance	200

* Tested by the response in the crops of pigeons (Riddle test).

† Tested by vaginal smear in castrated rats (Allen-Doisy test).

‡ Fluid was removed from cysts of both breasts of this patient. The milky fluid from the right breast contained 6,000 rat units of estrogenic substance per liter. The yellow turbid fluid from the left breast did not contain any estrogenic substance.

Although the experiments of Loeb and Lacassagne would seem to indicate that estrin may play a rôle in the formation of carcinoma of the breast in mice, bio-assays for estrin in the tissue of mammary carcinoma removed from patients have yielded no strikingly positive results. The majority of cases assayed to date have proved negative (Table IV).

ENDOCRINE STUDIES IN UTERINE MYOMATA.—Myomata of the uterus and endometrial hyperplasia are associated with ovarian tumors of the granulosa cell type. In over 125 granulosa cell tumors of the ovary appearing in the literature, endometrial hyperplasia, thickening of the musculature or the occurrence of adenomata or adenomyomata have been common (Stefancsik[29]). In such cases increased blood levels of estrin and an increased urinary output of the same hormone have been reported. When estrin has been injected in rabbits it has been noted frequently that the musculature of the uterine horns has been doubled or trebled in thickness as a result. Bearing this in mind we have assayed myomata of the uterus for estrin (Table V). The high concentration (11,000 rat units per kilo) of prolan, or a pituitary-like sex hormone present in some of these tumors, would seem to indicate that this hormone may play a synergistic rôle in stimulating the musculature.

TABLE IV

BIO-ASSAYS OF ESTRIN, PROGESTIN, AND GONADOTROPIC SUBSTANCE IN CANCER OF THE BREAST

Patient	Age	Diagnosis	Hormone Assayed	Result of Bio-Assay (Rat Units per Kilo)
M. H.....	50	Adenocarcinoma	Prolan	Negative
			Estrin	Negative
R. L.....	43	Scirrhus	Progestin	Negative
C. C.....	50	Scirrhus	Progestin	Negative
			Estrin	Negative
L. L.....	65	Colloid	Estrin	Negative
M. L.*.....	37	Scirrhus	Estrin	Negative
G. K.....	36	Scirrhus	Estrin	2,500
			Prolan	Negative
D. B.....	51	Infiltrating duct ca.	Estrin	Negative
E. S.†.....	32	Grade IV carcinoma	Estrin	850
			Prolan	7,000
B. B.†.....	47	Grade IV carcinoma	Estrin	850
			Prolan	Negative
M. C.†.....	65	Papillary	Estrin	Negative
			Prolan	Negative
CAR.†.....	45	Papillary	Estrin	Negative‡
			Prolan	Negative
C. C.....		Papillary	Estrin	2,500
M.....		Papillary	Prolan	6,000

* Virginal hypertrophy with cancer. Blood positive for estrin on seventh day of period.
† Checked by Doctor Morrell.
‡ Positive for estrin on direct injection of tissue.

TABLE V

BIO-ASSAYS OF ESTRIN AND GONADOTROPIC SUBSTANCE OF MYOMATA OF THE UTERUS

Patient	Estrin (Rat Units per Kilo)	Gonadotropic Substance (Rat Units per Kilo)
Control normal Premenstrual uterus..	3,000	
P.....	4,000	4,500
J.....	1,000	11,000
G.....	Negative	Negative
T.....	Negative	Negative
R.....	600	11,000
D.....	200	—*
O.....	30,000	—*
J.....	600	—
B.....	600	—
O.....	600	—
B.....	250	—
K.....	Negative	—
J.....	Negative	—

* Not done.

Studies of the Occurrence of Parathormone in Giant Cell Tumors.—Erdheim,[12] in 1907 called attention to changes in the bones associated with parathyroid adenoma. Mandl,[27] in 1926, conclusively proved the endocrine basis underlying tumors and cysts found in von Recklinghausen's disease. He removed a parathyroid adenoma and obtained clinical improvement in a case of multiple osteitis fibrosa cystica. Since this date many contributions (Hunter,[20] Ballin and Morse,[3] etc.) have emphasized the endocrinological factor in this disease.

While hyperparathyroidism, with an elevation of blood calcium is an etiological factor in von Recklinghausen's disease, no parathyroid changes nor increase of blood levels of calcium or parathormone have been found in solitary giant cell tumor or solitary bone cysts. Repeated determinations of calcium and phosphorus in the blood serum of patients with solitary giant cell tumors and bone cysts have been made in this clinic and elsewhere. However, nothing has been found which would indicate a state of hyperparathyroidism.

However, giant cell tumors may occasionally occur in the latter third of pregnancy. We have seen several instances in the long bones. Epulis of the alveolar margin, developing during pregnancy, is a well recognized clinical variety of giant cell tumor. Hamilton[18] has demonstrated that the parathormone output is increased during pregnancy. In several instances of giant cell tumor occurring in patients between 39 and 60 years of age, the long bones in the unaffected extremity showed a visible epiphyseal line. These findings suggest a possible disturbance in bone metabolism as a factor in the development of solitary giant cell tumor. The possibility that a localized increase in the concentration of parathormone may occur at the site where the tumor develops has led us to attempt the bio-assay of solitary giant cell tumor for parathormone. Two cases treated by preoperative irradiation gave negative results.

Three additional benign giant cell tumors treated by primary curettement gave positive bio-assays for parathormone. Dogs weighing approximately 10 kilograms were injected with the equivalent of 4 to 8 grams of tumor tissue, and showed an increase of 18 to 22 per cent in their blood calcium 15 hours after the initial injection.

The tumor tissue was extracted in 5 per cent hydrochloric acid in a boiling water bath for 45 minutes (Collip[8]). The digested material was diluted with four parts of hot water and subsequently chilled to remove the fat. The liquid was then raised to a P_H 8 to 9 with sodium hydroxide. Five per cent hydrochloric acid was added slowly until a precipitate occurred (at about P_H 5.5). The material was then filtered, saving both filtrate and precipitate. The precipitate was again made alkaline and again precipitated as above. This process was repeated three times. The hormone was then recovered from the free filtrate by making the solution acid to congo red and then

saturating with sodium chloride. The flocculent, active material was then separated and dissolved in weak sodium hydroxide, and the liquid adjusted to P_H 4.8. The precipitate was centrifuged and the supernatant liquid was treated as above. The precipitates were then added together and dissolved in a weak hydrochloric acid solution at P_H 3. This active solution was injected into the dog after being neutralized just before injection.

Table VI shows the results of assays on parathormone in giant cell tumors and in a case of osteolytic sarcoma, occurring three and one-half years after curettage and irradiation for benign giant cell tumor. In addition, assays for growth hormone and gonadotropic substance in benign osteochondroma and osteogenic sarcoma are recorded.

TABLE VI

BIO-ASSAY OF PARATHORMONE, GROWTH AND GONADOTROPIC HORMONE IN BONE TUMORS

Patient	Diagnosis	Hormone Assayed	Result	Units per Kilo Dry Weight
J. C. B..... 2–15–35	Giant cell tumor (irradiated)	Parathormone	Negative	0
Monroe.... 9–11–35	Giant cell tumor (irradiated)	Parathormone	Negative	0
Henry..... 1–21–36	Giant cell tumor	Parathormone	Positive	25,000
Irwin..... 3–11–36	Giant cell tumor	Parathormone	Positive	14,000
Mentzner.. 7–29–36	Giant cell tumor	Parathormone	Positive	14,000
Brown..... 10–11–34	Sclerosing osteogenic sarcoma (irradiated)	Growth hormone	Positive	65
		Gonadotropic hormone	Positive	4,500
Ewing..... 3–11–36	Osteolytic sarcoma	Parathormone	Positive	25,000
Harper.... 4–10–35	Osteochondroma	Growth hormone	Negative	0

CONCLUSIONS

No conclusions are to be drawn from the results of these bio-assays regarding the causal relationship between the hormones and tumor formation. In certain benign lesions of the breast, such as cystic disease and fibroadenomata, these hormones may be recovered in a high percentage of cases, while they are inconstant, or absent, in carcinoma of the breast. On the other hand, the methods in use for extracting hormones from tissue do not neces-

sarily yield the total amount of active substance from the tissue. It has been shown, particularly in regard to estrogenic substances, that various methods of extraction will yield different quantitative results or a variety of active fractions. Preliminary hydrolysis of the tissue with hydrochloric acid or injection of an emulsion of freshly ground tissue (when not too toxic) may give higher values than the routine method of hot alcoholic extraction.

The exact composition of the hormone, its source and its ultimate fate in the body, often remains in doubt after repeated assay of the tissues, blood and urine in a given case. While the structural formula for the female sex hormone is known (ketohydroxyestrin and trihydroxyestrin), estrogenic activity has been demonstrated for a series of condensed ring compounds of similar structures which are closely related to the sterols and bile acids (Cook, Dodds and others[9]). Hence no conclusion regarding the chemical identity of the substance can be drawn from bio-assay. A varied group of substances likewise may produce the Aschheim-Zondek[2] reaction.

The above assays are merely recorded as facts and any attempt at interpretation must await the results of further investigation. The presence of these endocrine substances in new growths would seem, however, to have significance in the physiology of the growth and to explain more satisfactorily, than any theory yet advanced, why tissues in a new growth reproduce themselves locally, and in metastases.

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Discussion.—Dr. Charles Carroll Lund (Boston, Mass.).—It is noteworthy to have as busy a surgeon as Doctor Lewis take an interest in chemistry and hormones, and I think it has an important bearing on the surgery of the future.

It might be of interest to know that in the recent general examination at the Harvard Medical School, the men were given great latitude in the choice of writing a paper, in a four hour test. They were given three hours of the four to write on any subject in medicine they wanted to, in which there had been recent advances. Three-quarters of the class elected some phase of endocrinology. It is obvious, therefore, that this is one of the fields the present student is interested in.

Doctor Lewis, wisely, does not draw any conclusions from his observations. The field is opening up so rapidly that the first thing of importance is to collect relevant data, and certainly the essayist has made noteworthy advances in the subjects considered.

There has been considerable discussion concerning these hormones being important as an etiologic factor of breast cancer. Gardner and Allen in New Haven, and others, have shown that there are at least three hormones

important in the development of the breast, the gonadotropic, estrin, and progestin, and in addition there is the prolactin, which is important in secretion. I think the conservative point of view to take about the animal experiments of Lacassagne and others, at present, is not to attribute to estrin, or any other hormone, the cause of cancer in male breasts or in breasts of castrated animals, but simply to consider that if you are going to have cancer in a breast you have to have breast tissue. On a quantitative basis, roughly, in the human male and female, you get about as much cancer in the male breast per gram of tissue as you do in the female breast. So that it may not be hyperestrinism, or any endocrine deficiency, but simply the number of cells that are exposed by time to other processes which cause cancer of the breast.

TRANSURETHRAL RESECTION

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The idea of attempting to overcome obstructions at the bladder neck by means of transurethral manipulations is not new. The historical side of this subject has been presented by various authors during the past few years and those interested in this phase of the subject may refer to these various publications.[1, 2]

With the development of a new armamentarium for the non-surgical relief of bladder neck obstruction, widespread interest in electro-resection has been manifested both by the medical profession and the public. No surgical subject in urology, since the fulguration treatment of bladder tumors by Beer,[3] has received as much attention and discussion as has resection.

From a review of recent publications and discussions it is evident that definite ideas are beginning to crystallize regarding the selection of cases suitable for resection, its value and limitations, as well as the results obtained.

The statement seems justified that three different points of view exist today regarding the selection of cases suitable for resection.

(1) With the introduction of any new procedure in our armamentarium one finds a group of enthusiasts who take up the new procedure with great eagerness and in this particular instance believe that this method will replace, or even has already completely replaced, surgical prostatectomy.

(2) On the other hand, one finds another group who are just as decided in their views as the first group and who believe that surgical prostatectomy for various reasons is still the method of election.

(3) In the third group one finds urologists who are of the opinion that there is a definite place in our armamentarium for both transurethral resection and surgical prostatectomy. These men willingly admit without controversy that resection is the method of choice in the treatment of bars and contractures, small middle lobes, and, in some of the cases, of small lateral lobes, but that large hypertrophies should be treated by surgical prostatectomy. In other words, this group is of the opinion that one should make a rather careful selection of cases and not fit every patient to any one form of treatment.

It is at this point that there will naturally arise differences of opinion regarding the procedure to be employed in a given case. One surgeon may express his opinion that the prostate is too large to be resected and that a prostatectomy should be performed; whereas, another will state that resection is possible and will carry it out.

My experience in a relatively large number of patients has coincided with the latter opinion; and I think that the recommendation for resection or prostatectomy may be influenced by the personal preference of the surgeon.

I have performed many resections, without difficulty of any kind, in patients who were told that this procedure was impossible. It is interesting to note the large number of doctors who come in for resection, despite the fact that they were told they needed open surgery.

On one point, however, there is very definite agreement, namely, in the treatment of carcinoma all agree that transurethral resection is the method of election combined with radium or deep roentgen therapy.

In trying to determine in my own mind the value and limitations of resection I have, as far as possible, approached this problem with an open mind and without prejudice.

My own position in this discussion can best be summarized by saying that during the past 51 months I have performed only one surgical prostatectomy and that during the same time I have refused resection to one patient only. This patient, a bad cardiac, was given suprapubic drainage until his cardiac function improved so that resection could be carried out. It is evident, therefore, that there has been no selection of cases, irrespective of the size of the prostate, the nature of the enlargement, or the physical condition of the patient.

From my experience in performing transurethral resections several very definite impressions have been gained; and while these views may not coincide with the views of others, I wish to present them at this time, realizing that perhaps some of you will not agree with me.

There is no doubt of the fact that the period of hospitalization is much shorter with resection than it is with surgical prostatectomy, and while this fact may not be of much importance to a small percentage of patients, I believe, all things being equal, it is an economic factor of enough importance to demand attention. Not only does this apply to the private patient, but also to the ward patient, as a result of which many more patients can be treated in a given period of time, hence making our ward services available to a larger number. The average stay in the hospital was seven days except when a preliminary suprapubic cystostomy was required, and in these cases the average stay was 20 days.

The number of patients in whom preliminary suprapubic cystostomy was employed became less and less as time went on. During the past three months only one preliminary cystostomy was done. On the other hand, I believe a cystostomy is indicated when catheter drainage fails because of chills, fever, pain, or bleeding, or when small stones with severe infection are present, or large stones.

Based on my experience with resection, I am convinced of the fact that resection has made it possible for a large number of patients to obtain relief from their prostatic condition who, because of serious coexisting disease in other important organs, were very poor surgical risks and, as a matter of fact, were refused prostatectomy elsewhere. It seems justifiable to state that if this procedure is of necessity the method of choice in the patient who is a very poor risk and hence cannot undergo a surgical prostatectomy with safety, the assumption that it should be made available to every patient who is a good risk is unquestionable.

It is not an uncommon experience to have a patient sent in by his physician with the request that a resection be performed because the patient is not a good risk for prostatectomy. The following instance is a good example of this type of case.

Case 1.—A. G., aged 76, admitted to the Presbyterian Hospital, March 1, 1936.

Present Complaint.—Frequency of urination for five years with gradually increasing severity; nocturia that has gradually increased, for three years. The day and night frequency accompanied with a great deal of pain on urination. During the past year there has been a good deal of difficulty in starting the stream; it was necessary to strain a long time before the urine would flow. Urgency and imperative urination during the past three weeks.

Physical Examination.—Pupils equal and reacted somewhat slowly to light. Numerous coarse and squeaking râles in the lungs; breath sounds at both bases tubular in character. The apex heart beat was not located; the sounds, of very poor quality. Abdomen: marked diastasis of the recti and a double inguinal hernia. Bladder dulness reached to the umbilicus. Liver palpable one inch below the costal arch. Rectal examination: a three plus enlargement of the prostate that was smooth and firm. Seminal vesicles could not be felt. The edema of the feet and ankles and legs extended almost to the head of the fibula. Lips and finger tips, cyanotic.

Laboratory Data.—Blood: red cells, 4,250,000; white cells, 10,800; hemoglobin, 74 per cent. Blood pressure: systolic, 170; diastolic, 70. Urine: albumin, 3 Mm.; sugar, 0; blood, +; leukocytes, 5,000. The colon bacillus was cultured from the urine. Blood chemistry: urea nitrogen, 14.3; creatinine, 1.2; nonprotein nitrogen, 33.2.

Roentgenologic Examination.—A marked diverticulosis of the colon and a bilateral hernia of the diaphragm. Multiple stones in the bladder. A small, contracted bladder with multiple cellules and small diverticula in the cystogram.

Indwelling catheter drainage was carried out for eight days.

Diagnosis.—Bilateral diaphragmatic hernia; diverticulosis of colon; bronchial asthma and emphysema; bundle branch block; myocardial damage; hypertension; arteriosclerosis; benign hypertrophy of the prostate; vesical calculi; chronic cystitis; bilateral inguinal hernia; diverticula of bladder, and hydrocele.

Operations.—On March 9, 1936, under sacral anesthesia, a transurethral resection was performed and a very large middle lobe and two rather large lateral lobes were removed. On March 16, 1936, under sacral anesthesia, a litholapaxy was performed. The patient had an uneventful convalescence. Discharged from the hospital, March 22, 1936.

Another interesting observation noted was the fact that the number of cardiac cases seeking relief showed a very definite increase. Whereas, in the days of surgical prostatectomy about 35.8 per cent of the patients suffered from cardiovascular lesions, a recent study of my resection cases showed 64.3 per cent, an increase of 28.5 per cent. This may be due to the fact that a more careful study of each case was made and hence more heart lesions were found than formerly. An electrocardiogram was made in each case prior to resection, and furthermore there is no doubt in my mind of the fact that more cardiacs were sent in for resection than were sent in for open prostatectomy because of the lower mortality rate.

Many of the patients with hypertension have been sent in with the request that a resection and not a prostatectomy be performed. The highest systolic pressure found was 260, and a resection was performed without the slightest difficulty.

The following case is presented as an example of a patient with a severe heart lesion.

Case 2.—M. C., aged 75, admitted to the Presbyterian Hospital, November 26, 1935.

Present Complaint.—Complete retention for ten days during which time he has been catheterized three times a day. There is some mental confusion.

Physical Examination.—A fairly well preserved elderly man. Head and neck, negative. Lungs, considerable wheezing at the left base posteriorly and moist râles. Heart, enlarged; irregular in rhythm; the sounds of poor quality; pulse, that of an auricular fibrillation. Abdomen, negative. Rectal examination: a two plus enlargement of the prostate that was smooth and regular.

Laboratory Data.—Blood: red cells, 4,350,000; white cells, 7,810; hemoglobin, 88 per cent. Blood pressure: systolic, 142; diastolic, 60. Urine: sugar, 0; albumin, 0; blood, 0; red blood cells, 0; white blood cells, 140; casts, 0. Blood chemistry: urea nitrogen, 14.8; creatinine, 1.4; nonprotein nitrogen, 32. Phthalein test: a total of 62 per cent of the dye eliminated in one and one-half hours.

Electrocardiogram.—Auricular flutter; ventricular extrasystoles; myocardial damage.

Roentgenologic Examination.—Negative for stone. A small bladder with trabeculated margin and some out-pouching from the left toward the base in the cystogram.

After three weeks of indwelling catheter drainage, bed rest and heart medication, the patient showed great improvement.

Diagnosis.—Coronary disease; auricular fibrillation and flutter; cardiac decompensation; cardiac hypertrophy and dilatation; myocardial damage; chronic bronchitis, and benign prostatic hypertrophy.

Operation.—On December 19, 1935, under sacral anesthesia, a transurethral resection was performed. The patient developed postoperative bleeding on January 6, 1936, three weeks after the resection. Two bleeding vessels were found; they were fulgurated and the bleeding promptly stopped. Patient discharged from the hospital January 17, 1936.

The importance of a careful study of the renal function requires no emphasis at this time, nor is it necessary to call attention to the fact that resection should never be performed until the renal function, if impaired, has been restored to normal or at least until it has become stabilized.

In the largest number of cases the improvement in renal function with treatment is rapid, as judged by the results of the functional tests as well as by the patient's general condition. In this group of patients the problem of determining the most opportune time for resection offers no serious problem.

I wish, however, to discuss the group of cases with badly impaired renal function in which the response to treatment was very slow and the improvement hardly perceptible, and the functional tests remained fixed at a high, or relatively high, level. At times these levels were very high so that the danger of renal insufficiency was an opposing factor in case the patient was to be operated upon. It is in this group that the decision to operate or not to operate is very difficult and at times greatly taxes one's clinical judgment. There is no doubt in my mind that in just this type of case transurethral resection should be the operation of choice and certainly carries with it less risk than does surgical prostatectomy. As an illustration of this type the following case is presented.

Case 3.—C. E., aged 57, referred by Dr. J. B. Herrick, was admitted to the Presbyterian Hospital, January 10, 1936.

Present Complaint.—Frequency of urination, urgency, burning, dribbling, and noc-

turia. These symptoms had been present for ten years. Patient has had pain in the epigastrium for ten years, nausea and vomiting for several months, and very tarry stools for several days.

Physical Examination.—Moderately well nourished; exceedingly pale and sallow. Head, neck and lungs, negative. Heart enlarged to the left; a systolic murmur at the apex. Examination of the abdomen showed a large tumor mass extending four finger-breadth above the umbilicus. Liver, palpable. Rectal examination: a one plus enlargement of the prostate, smooth, with no signs of carcinoma.

Laboratory Data.—Blood: red cells, 2,520,000; leukocytes, 9,850; hemoglobin, 44 per cent. Blood pressure: systolic, 150; diastolic, 96. Urine: albumin, 0; sugar, 0; blood, 0; leukocytes, 0. Blood in the stool. Blood chemistry: urea nitrogen, 105.6; creatinine, 6.3. Phthalein test: only a trace of dye excreted in one and one-half hours (Table I).

Roentgenologic Examination.—Negative for stone. A set of intravenous pyelograms showed no visualization. Further studies by Doctor Herrick established the fact that the patient had a duodenal ulcer and a polyp in the pyloric antrum.

A catheter was passed into the bladder and gradual decompression instituted. This required 21 days. The patient was given three blood transfusions, each 500 cc. of whole blood.

TABLE I

SUMMARY OF THE FUNCTIONAL TESTS IN CASE 3

				Phthalein Test	
Date	Urea N.	Creatinine	N.P.N.	Appearance Time	% in 1½ hrs.
1–11–36	105.6	6.3	.....		
1–13				45 min.	Trace
1–16	69.8	3.7	74.4		
1–23	83.3	2.9	102.6	42 min.	Trace
1–29	102.5	2.6	123.0		
2– 6	129.0	2.9	142.9		
2–12	91.8	2.7	125.8		
2–17	90.1	2.9	105.3	55 min.	Trace
2–24	58.8	2.7	74.1		
3– 2	55.1	2.5	86.7		
3– 4	55.6	2.5	72.7		
3– 8				10 min.	11%
3– 9	56.2	2.3	92.2		
3–18	59.2	2.4	76.2		
3–21				19 min.	30%
3–23	54.0	2.9	70.2		
3–30	54.4	2.6	61.5		
3–31				12 min.	26%

Diagnosis.—Polyp of stomach; ulcer of duodenum; marked secondary anemia; hypertension; severe hemorrhage from intestinal tract; benign prostatic hypertrophy; chronic urinary retention; marked renal insufficiency; bilateral hydronephrotic atrophy of kidneys.

Operation.—On April 1, 1936, a transurethral resection, under sacral anesthesia, was performed. The patient had an uneventful convalescence. Discharged from the hospital on the fifteenth postoperative day.

I cannot agree with those who believe that because resection carries with it a much lower mortality rate, it is a relatively simple procedure, and that careful, detailed preoperative study and preparation can be dispensed with

and resection instituted at once. It seems to me that this is both poor teaching and poor practice. Because of the large number of cases that I have seen with serious organic disease,[4] not only in the upper urinary tract but in other organs, I believe it is absolutely necessary to obtain a careful history, as well as make a complete physical examination in each case and carry out the necessary preoperative treatment.

As my experience with resection has widened I have operated upon a certain group of cases without preliminary catheter drainage. Heretofore all patients who had four ounces or more of residual urine were given preliminary catheter drainage. At the present time no preliminary drainage is em-

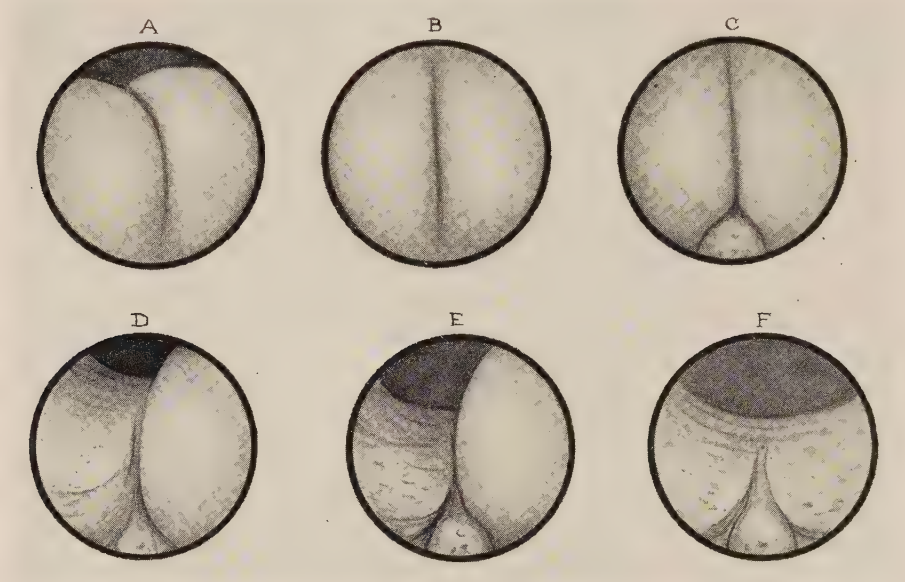

FIG. 1.—(A) Showing a small view of the bladder cavity and the presence of two large lateral lobes. (B) Showing the presence of the lateral lobes and the very markedly narrowed urethra. (C) Showing the lateral lobes and the verumontanum. (D) Partial removal of the right lobe. (E) Complete removal of the right lobe. (F) Complete removal of both lobes. The verumontanum is intact.

ployed in a patient in whom the residual urine does not exceed six ounces, provided the urine is clear, the renal function normal, and the heart and lungs are in good condition.

Preliminary cystoscopy is no longer carried out as a routine; once the diagnosis of prostatic obstruction has been made the type of enlargement is determined at the time the resection is performed. The exception to this rule is in case the history is not typical for prostatic obstruction, there is a history of one or more attacks of hematuria, and the cystogram shows a filling defect.

After the sheath has been passed into the bladder the examining telescope is introduced and the bladder cavity examined for tumors and stones. A search for diverticula is also made and finally the configuration of the bladder orifice is determined (Fig. 1 A, B and C).

The bladder orifice is next examined with the retrograde lens, and a good deal of information is obtained regarding the size of middle lobe and bars (Figs. 2 A and B and 3 A). Hypertrophy of the interureteric ligament and the resulting pouch behind it can best be demonstrated with the retrograde lens. When these conditions are marked I always resect the hypertrophied interureteric ligament.

The third and last step is the examination of the prostatic urethra to determine the size and extent of the intra-urethral enlargement and its relationship to the verumontanum (Fig. 1 C).

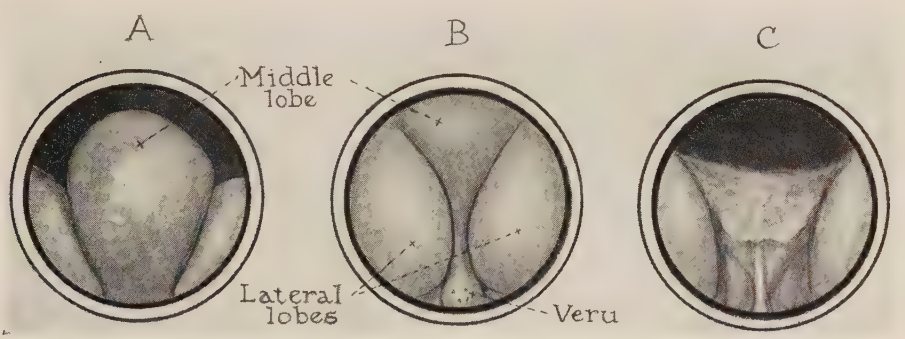

FIG. 2.—(A) Showing the presence of a large middle lobe. (B) View showing the lateral lobes, the middle lobe, and the verumontanum. (C) Beginning resection of the middle lobe.

POSTOPERATIVE REACTIONS.—The general opinion seems to prevail that following resection there are fewer postoperative complications and that when they do occur they are not so severe and are relatively much shorter in duration. I can subscribe to these views.

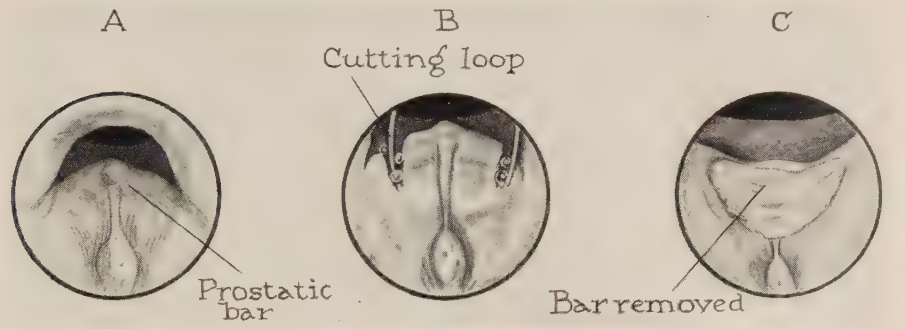

FIG. 3.—(A) Showing the presence of a bar. (B) Removal of bar. (C) The hypertrophied interureteric ligament comes into view.

MULTIPLE RESECTIONS.—Objections to this method have been made because of the fact that a second or even a third resection is necessary. This, I do not believe, is sound reasoning and requires some qualification. In this series of resections a second one was performed in 10.6 per cent of the cases, and a third one in 3.7 per cent. A study of this group shows that the cases can be divided into, and discussed under, three headings.

(1) In the cases of carcinoma a second resection is necessary when the carcinoma, because of its recurrence or continued growth after the resection, again produces obstruction. I believe that two resections for carcinoma are preferable to one suprapubic cystostomy and a permanent suprapubic drain or catheter.

(2) In this group are the patients with large benign hypertrophies in whom, it can readily be understood, the obstruction cannot be removed at one operation, because of the size of the growth. I always tell these patients that a second resection may be necessary.

(3) In the remaining cases in this group are the patients in whom not enough tissue was removed at the first resection. This occurred with some frequency in the early cases, but with experience a second resection became less and less frequently necessary.

Hemorrhage.—Before the technic of resection was completely mastered, hemorrhage at the time of operation was at times a serious problem, but with experience this untoward occurrence becomes less and less of a problem, although occasionally a serious hemorrhage occurs no matter how careful one may be. Careful search for the bleeding point should be made and its immediate control effected. I endeavor to have the patient return to his room with the wash water free of blood, that is, clear or faintly tinged. Each bleeding point should be isolated and fulgurated before proceeding with the resection, and, above all, blind indiscriminate coagulation is to be avoided. I believe that late secondary bleeding and the persistence of infection are due in some cases to indiscriminate widespread and deep coagulation.

Secondary bleeding occurring on the tenth to the fourteenth day was present in 19 cases but was never severe. Clots, when present, were evacuated with the Bigelow pump and the bladder washed out with a warm solution of silver nitrate. In most of the cases this controlled the bleeding, but when this procedure failed the resectoscope was introduced, and the bleeding point fulgurated or excised.

Late hemorrhage occurred in 14 cases, at periods varying from three months to two years after the resection. In the cases that I have had an occasion to reëxamine, enlarged vessels around the bladder orifice and in the prostatic urethra were found. These were destroyed by fulguration. An interesting fact is that these late hemorrhages occurred in patients with very large prostates.

In one instance in a patient aged 80, hemorrhage occurred 26 months after resection. A suprapubic cystostomy was performed (elsewhere) in order to control the bleeding. The patient was unable to withstand even this simple procedure and died.

Epididymitis.—The incidence of epididymitis has diminished as experience with resection has grown. Of the first 117 cases 15 per cent had this complication, and a routine vasectomy was done, an operation no longer employed, as this complication is very rare.

Results.—In order to determine the results obtained with resection, two methods of investigation were used: (1) In so far as it was possible, every

attempt was made to have the patients return to the office or hospital for a personal examination. (2) Many of the patients who lived out of town could not, or would not, travel to the city for personal interviews and examinations, hence it was necessary to resort to a questionnaire.

When the obstruction had been completely relieved and there were no complicating conditions present, the symptoms rapidly subsided, the pyuria disappeared, and the urine became clear. It might be well, at this time, to mention the fact that in some of the patients in whom the pyuria persisted, without symptoms and without residual urine, and in whom no local treatment was instituted, examination at the end of from three to six months

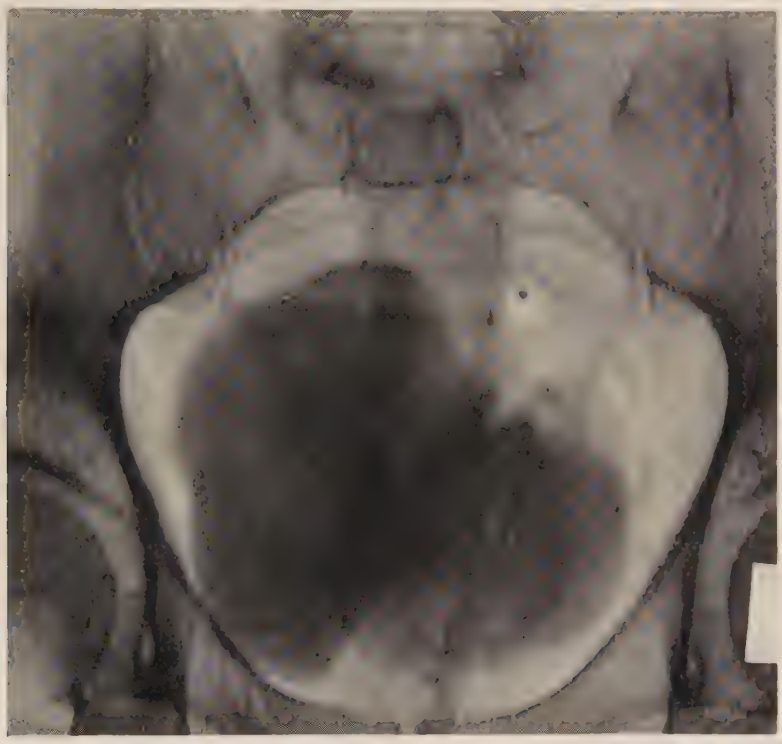

FIG. 4.—Showing a diverticulum much larger than the bladder.

showed a complete disappearance of the pyuria. In other words, in some of the cases a certain elapse of time was necessary before the urine became clear. This can readily be understood because cystoscopic examination in some of these cases still showed the presence of white sloughs at the end of 11 weeks. Aside from the slow healing in some of the cases there were other causes for the persistence of the pyuria, among which may be mentioned:

(1) *The Presence of One or More Diverticula.*—In some cases one or more diverticula were responsible for the turbid urine, although the patient was symptom free; under these circumstances it was difficult to convince the patient that a diverticulectomy should be performed in order to clear up the turbid urine (Fig. 4).

(2) *The Presence of Large Hydronephrosis.*—In some of the cases of persistent pyuria, hydronephrosis was the contributing factor; even after the prostatic obstruction had been completely relieved, the hydronephrosis and hydro-ureter persisted, with stasis and infection, and hence pyuria. This cannot be reasonably charged as a failure of resection.

(3) *Infection in the Seminal Vesicles.*—Infection in the seminal vesicles as a cause of the persistence of symptoms and turbid urine following surgical prostatectomy is recognized by all urologists, and was stressed by Boyd[5] some years ago. If symptoms and turbid urine persist after a reasonable time and if infection is found in the seminal vesicles upon examination, then the use of heat per rectum, systematic massage, and instillations will result in completely alleviating the symptoms and clearing up the urine. It is well to bear in mind that even when the urine is clear infection in the vesicles may be the cause of symptoms.

(4) *The Formation of Stone.*—Calculus formation following resection has been rare in my experience, having occurred only four times; three in my own cases and once in a case resected elsewhere. After litholapaxy there was a complete disappearance of the pyuria and the symptoms.

(5) *Failure to Relieve the Obstruction.*—Naturally, failure to relieve the obstruction results in a continuation of the symptoms, as well as a persistence of the pyuria.

(6) *Indiscriminate, Widespread Coagulation in Controlling the Bleeding at the Time of Resection.*—This has already been considered under hemorrhage.

Residual Urine.—The objective in the treatment of the patient suffering from prostatic obstruction has, as its ultimate goal, the relief of the obstruction; whether or not this result has been attained can best be determined by the estimation of the residual urine. It is necessary to bear in mind that the amount of residual in a certain number of cases will gradually diminish. I have seen patients who, when discharged, still had an ounce or two of residual urine, and when examined six months later had only a few cubic centimeters. It is fair to assume, therefore, that in some of the patients mentioned in the table, smaller amounts of residual urine could be demonstrated if the opportunity of an examination were presented today.

I have had the opportunity of examining 273 patients for the purpose of determining the question of residual urine (Table II).

Table II

AMOUNTS OF RESIDUAL URINE IN 273 CASES FOLLOWING TRANSURETHRAL RESECTION

No residual urine	181 cases	(66.30%)
10–20 cc.	44 cases	(16.11%)
Total		(82.41%)
20–30 cc.	17 cases	(6.22%)
30–40 cc.	5 cases	(1.83%)
40–50 cc.	6 cases	(2.19%)
50 or more cc.	20 cases	(7.32%)

A detailed analysis of the 20 patients who had 50 or more cubic centimeters of residual urine is presented in Table III.

TABLE III

ANALYSIS OF CASES WITH HIGH RESIDUAL URINE

Diverticula of bladder	5 cases
Carcinoma of prostate	5 cases
Large hydronephrosis	2 cases
Bladder and prostatic calculi	2 cases
Periprostatitis	1 case
Second resection advised	2 cases
No return for check-up	3 cases

In some of these cases the persistence of turbid urine was due to changes which might be directly ascribed to the bladder neck obstruction and hence one cannot rightfully classify such cases as failures; for instance, in five cases the diverticulum was responsible for the persistence of turbid urine, and I am of the opinion that part of the residual urine can also be explained on this basis. In the cases of carcinoma of the prostate the carcinoma continued to grow and hence produced obstruction. In two cases the large hydronephrosis was responsible for turbid urine and a certain amount of residual. The two patients with bladder calculi were relieved of the turbidity of their urine and obstruction as previously mentioned under the subject of formation of stone following resection. In two cases sufficient tissue had obviously not been removed and the patient refused to have a further resection. And, finally, in three instances it was impossible to have the patients return for a check-up.

INCONTINENCE.—There has been no instance of complete incontinence in this series of 804 resections. Shortly after leaving the hospital a small number have had difficulty in holding urine. This was not due to incontinence, but to urgency and imperative urination. Continence was eventually recovered. In a few instances the patients would lose a few drops, but this was temporary and, as the infection cleared up, the condition rapidly disappeared. In two instances the patients' loss of a few drops was due, not to incontinence, but to stricture of the urethra.

In one instance, a recent case, lack of control was rather marked and annoying during the daytime, but at night the control was perfect. The patient was given ephedrine, and at examination, three days ago, showed great improvement.

In a second case, when last seen three months ago, the patient had perfect control at night and during the day except when he assumed certain positions, and then a few drops would escape.

URETHRAL STRICTURE.—Whether or not stricture formation will occur in a sufficiently large number of cases, to justify the statement that resection produces stricture, must be determined with the passage of time, and the same should be said regarding the question of whether or not it will occur more frequently with resection than it did with surgical prostatectomy. Doubt has recently been expressed as to whether or not resection produces a stricture,

it being the opinion of some that when stricture is found following resection the stricture was present before operation.

Sexual Function.—So far as the sexual function was concerned, there has been no decided change. In the majority of the cases there has been no change, whereas, in some, with an improvement in the general condition, there has been an improvement in the sexual function. In only a few cases has there been a slight impairment of function.

Recurrence.—A good deal of discussion has centered around the question of whether or not recurrence will take place following transurethral resection. This question cannot be definitely answered at this time. It is a well known fact that recurrences have taken place following both suprapubic and perineal prostatectomy. One of the characteristics of recurrence following surgical prostatectomy is the fact that the interval between the removal of the prostate and the development of symptoms which demand further prostatectomy averages about eleven and one-half years.[6] It stands to reason that if recurrences are going to occur, the time since the introduction of resection has not been long enough for them to develop. On the other hand, should recurrences occur after a period of five or ten years, a second transurethral resection should be a perfectly simple procedure.

Mortality.—The mortality rate has fluctuated from time to time, depending in part upon the type of cases and whether or not one wishes to refuse resection to many of the so called "bad risk" cases, which I have not done. My records show that I have performed 184 resections with but one death—a mortality of .54 per cent. Recently a large number of "bad risk" cases have presented themselves and the mortality in 804 resections was 3.9 per cent.

If resection can relieve both obstruction and symptoms, and also has to its credit a shorter period of hospitalization, a lower mortality rate, and fewer postoperative complications than in the cases in which prostatectomy is performed, it obviously has merit and should find its proper place as a surgical procedure.

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Discussion.—Dr. Edwin Beer (New York, N. Y.).—A certain amount of exaggeration and overenthusiasm may be necessary to call attention to, and to put over, a new surgical procedure. Whether this is due to the natural lethargy of a large part of the medical profession, I do not know. As a result of this overswing of the pendulum, at times harm is done, but this is usually only a temporary state. Eventually the pendulum swings back, a normal equilibrium is established, and the exact position of such a new procedure as Doctor Kretschmer has so clearly presented to us, is recognized. This cor-

rection of the overenthusiasm is already manifest in the literature, and before long we shall recognize clearly the possibilities of this new method of attack, and at the same time its limitations and dangers.

About 1909, Hugh Young started the transurethral removal of fibrous obstructions at the bladder neck, after the various attempts of European surgeons along these lines, during the latter half of the last century, had given way to the operation of prostatectomy. One year later the profession became acquainted with the surgical use of the high frequency current in the bladder, and its possible therapeutic value in instances of bladder papillomata, tuberculous ulcers of the bladder and prostatic adenomata. Since those days, the instrumental advances and the modalities of the currents have been much improved and modified, so that at present we have a variety of instruments for cutting out pieces of the bladder neck and of the prostate, either by punch procedures followed by cauterization, or by resectoscopes with loops of one sort or another, activated by the high frequency cutting current of such oscillations and intensity as to cut living tissue under water. These changes are in the nature of a great and fundamental advance.

The three types of cases in which these new methods are of use are as follows:

(1) Contractures of the neck, which in years past were excised by wedge excision from above through the bladder. These can be cut with a point or loop electrode with complete relief of symptoms, using an ordinary operating cystoscope or a resectoscope. This is a great improvement over the cold punch operation or the suprapubic excision. The latter procedure or approach, however, may be useful when the exact physical status of the neck of the bladder is not accurately visualized.

(2) Obstructions caused by carcinoma of the prostate also lend themselves very satisfactorily to these transurethral resections, which may be repeated as new obstructions develop. The pathologic tissue removed during these operations is often helpful in establishing the correct diagnosis.

(3) When it comes to the ordinary and usual type of prostatic hypertrophy or adenoma, the field of transurethral resections has no limitations for some enthusiasts and very definite limitations for others. In this group of patients, there is a subgroup with mild symptoms of frequency and questionable obstruction, associated with a very small amount of residual urine and a very moderately enlarged prostate, upon whom no experienced surgeon would have felt justified in performing a prostatectomy. These patients present a definite potential field for transurethral resections, and may represent a large proportion of the total in most published series of resections. The frequency, and perhaps infection, do not regularly disappear, but may be made worse following these excisions of some prostatic tissue, despite reduction of residual urine. These cases are definite beginning prostatic adenomata and have nothing to do with those cases which have been resected prophylactically before any definite complaint has developed. It is doubtful whether resection affords these patients the expected relief with any regularity. If the subjective complaints are well developed, one is justified in attempting to give relief by resection, even though it may be only temporary and incomplete. Correct diagnosis here, as always, is essential.

In addition to these mild prostatics who frequently are worse off after a resection, there are two other groups of definite prostatic adenomata that must be discussed: (a) the moderately large, bilobe, trilobe or single lobe prostate, and (b) the very large, more or less trilobe or bilobe hypertrophy. In those localities where no pressure is brought to bear upon the surgeon by competitors or by the patient or his friends, the opinion is gradually devel-

oping that the last mentioned group of very large, succulent adenomata should be treated radically and definitively by prostatectomy or enucleation of the adenomata, either by the suprapublic or the perineal approach. This attitude seems to be based on a great many factors, without denying that if the "resectionist" is patient he can whittle away a fair amount of the largest prostates in one or more sessions.

The operation of suprapubic prostatectomy in two stages has gradually assumed the status of an almost minor procedure, with a low mortality, 6 plus per cent, usually incidental to cardiovascular disturbances, and with complete cure and no chance of recurrence, if properly performed. On the other hand, the transurethral approach in these cases frequently is followed by a whole group of new complications, such as primary or secondary hemorrhage into the bladder, requiring repeated evacuation of clots, invariably causing great discomfort, infection of the bladder and the more or less completely resected prostate base, which frequently, much more frequently than is reported, may turn the scales against the patient, not to mention the various instrumental damages and perforations that are reported by competent instrumentalists. In this group of cases, the prolongation of the necessary instrumental work and the various complications are liable to have a definitely higher mortality than follows a two stage suprapubic enucleation. This is our experience, and I believe if this type of case were alone considered, the data of other clinics would probably bear me out. The trouble with most statistical studies along these lines is that the mortality statistics are grouped covering the simple cases of contracture, the small, almost symptomless hypertrophies, the prophylactic excisions, perhaps incorrectly interpreted cystoscopically, and the moderate sized but definite prostatic adenomata, as well as the large hypertrophies.

In the moderately enlarged prostates, as opposed to the very large adenomata which weigh one or more ounces and are as large as one to three or more lemons, there is a definite field of usefulness for this method, even though repeated sessions may be required before the patient can void and be comfortable, and even though one may, as time goes on, see more and more of these partially removed adenomata redevelop and produce recurring symptoms, which is a rare sequela to a properly executed, complete enucleation. There is no doubt that in this group of moderately enlarged prostates one is justified, if the patient requests it, and especially if he is a poor surgical risk owing to poor renal function, coronary disease, diabetes, and so forth, in using transurethral resection. From my experience, I find a careful follow up of these resected cases leaves much to be desired, and perhaps in view of the ease with which transurethral resections can be done with our modern instruments, the technician is liable to be too enthusiastic, emphasizing the procedure rather than the result. Further experience will decide whether the life expectancy of resected patients is equal to, or as long as, that of patients who have had a total prostatectomy.

In traveling during the winter in the South, I have been impressed by the reports of large numbers of "resected" patients who require continued treatment, soundings and bladder irrigations, *etc.*, which practically no properly prostatectomized patient ever requires. There is no doubt that most resected cases void more readily and have much less residual than prior to operation, but large numbers are not absolutely and definitely cured, as they are after a complete enucleation of the adenoma by operation. Pyuria, frequency and dysuria often persist for months or permanently in every series in which a really careful follow up is maintained.

That transurethral resection of the prostate has become an unusually

popular procedure, without due selection of cases, cannot be denied. This is clearly evidenced by the fact that few clinics in the first 30 years of this century were able to report over 1,000 cases in whom prostatectomy had been indicated and performed, whereas in the last five years a number of clinics have reported over 500, and some even 1,000 to 2,000, cases that have been suitable for and required transurethral resection. Is the male in our race degenerating so rapidly, or is every male a potential victim of the resectionist? Even after broadening and extending the indications for treatment of prostatism by prostatic resection, it takes a lot of imagination to believe that this sudden increase in prostatics is not a product of poor, inadequate and slipshod diagnostic investigation. Naturally it follows that in these cases, without the usual imbalance caused by real prostatic adenomata, the mortality of resection must be low, the risk being almost negligible.

We must remember that we are doctors, and not be carried away with technical performances which savor occasionally of tricks. Undoubtedly the popularization of transurethral resection has done infinite damage to patients throughout the country, to mention only the reported great increase in the sale of penis clamps for postoperative incontinence. It may take some time before the fever abates, but from an adequate, carefully studied series of cases, not running into overwhelming thousands, I feel sure that the profession will decide, much as I have done in the above few words, as to the field and scope of transurethral resection.

Dr. William E. Lower (Cleveland, Ohio).—The procedure of transurethral resection of the bladder neck for obstruction has been revived in the last few years and, as Doctor Kretschmer has said, this is probably due to improvements in instruments and electrical apparatus. Not only are the median bars and the sclerotic obstructions resected now, but the use of transurethral resection is being extended to include a much wider field, especially in the treatment of prostatic hypertrophy.

I think one thing Doctor Kretschmer did not stress sufficiently is that transurethral resection is not a simple procedure. Technically, it is more difficult than prostatectomy and the convalescence following this operation is, in many cases, not without complications. If the operation is not well done, incontinence occurs with its associated annoyance.

In what cases should this procedure be used? Experience has taught us that its scope of usefulness is much wider than we formerly thought, but again, I want to emphasize the danger of broadcasting the simplicity of the operation. However, it is true that it carries with it a low mortality rate and, so far, the postoperative results in most cases have been very good. In approximately 500 resections which we have performed, the mortality rate has been 1.8 per cent and we have had a series of 225 cases without a death. These are not selected cases, but include those of all types, as well as many feeble and elderly persons.

I want to emphasize a few things Doctor Kretschmer pointed out. First, the shortening of the preoperative period without catheter drainage. We no longer put in a catheter or make a cystoscopic examination in these patients. If a patient comes in with a history of frequency and especially of nocturia, we plan to make a cystoscopic examination and prepare the patient for a resection. If, at the cystoscopic examination, we find that the obstruction is not suitable for resection, we treat the patient in another way. But if it is suitable for resection, we do it at the same time, thus avoiding catastrophes that sometimes follow the use of the indwelling catheter.

As we have gained wider experience, we have removed larger and larger

amounts of tissue. Another advantage of this operation is that a second or even a third operation may be performed if necessary, without any special risk.

Bleeding is becoming inconsequential. When the procedure was first used, bleeding occurred frequently, but in only one case have we had to open the bladder.

The incidence of epididymitis in the entire series is 6.8 per cent, and the average hospital stay is less than seven days; this economic factor is of considerable importance, especially in these times.

There is no doubt that this method is very practical and of far reaching importance, but its use must be limited to those who have had sufficient experience to perform it properly; otherwise, we are going to have many crippled old men and a high mortality rate from a method which, when handled properly, is an extremely valuable one.

Dr. Hugh Cabot (Rochester, Minn.).—In the first place, there are various methods of whittling out the prostate through the urethra. My notion is that the instruments will probably be greatly modified. But I insist we ought to discuss the problem without prejudice. Doctor Beer has perhaps a case to prove. I think he does chiefly prostatectomy. Doctor Kretschmer has a case to prove. He does chiefly resection. I have no case to prove.

Doctor Beer refers to the unfortunate results of resection. If we would tell the truth, we could load the statistics pretty handsomely with the unfortunate results of prostatectomy. The mortality of prostatectomy, which up to the last few years was in more than half of the cases in the hands of the occasional surgeon, has never the country over been less than 20 per cent, and is not now. Prostatectomy is an operation for experts, whether you use the suprapubic, transurethral, or the perineal method. I insist that we ought to confine our discussion to the results obtained by experts, not the results obtained by people who operate upon the patient not for prostatic obstruction but for a hundred dollars. That will cause damage, and does, in every field of surgery. That is not fair to the operation.

Six years ago, more than half the patients on my surgical list were prostatectomies. I have not done a prostatectomy in three years. There have been only three or four done in the last two years at the Mayo Clinic.

The age group is interesting. One thousand nine hundred twenty-two consecutive prostatectomies, done between 1924 and 1932, showed a 5.6 per cent incidence of patients over 75 years of age. The last 700 cases, up to January 1, 1936, show 15.7 per cent of the patients over 75.

It is frequently suggested that this operation is being performed chiefly for people who do not need it. I do not think that is true. We are operating upon sicker patients and with better results.

It has been the habit at the clinic, and it is not a bad one, to keep all the tissue which the surgeons remove. About a year ago I thought it would be interesting to take a series of 1,000 cases, to give a fair average of the gross amount of prostatic tissue removed by suprapubic prostatectomy, and compare it with the gross amount removed by transurethral resection. The amounts removed by resection are approaching those removed by prostatectomy. The group at the clinic doing resections now take out almost as much tissue on the average as was taken out by prostatectomy five years ago. It thus appears the transurethral method is becoming the method of prostatectomy. If we insist upon the same amount of skill for this method as is shown by the experts using the suprapubic or the perineal route, then resection has much to offer and will cover a large portion of the field.

Dr. Herman L. Kretschmer (Chicago, Ill.) closing.—I agree with Doctor Beer that a great deal of harm is done by enthusiasts, whether they be in the field of general surgery, urology, or internal medicine. I have tried to approach the problem of transurethral resection with an open mind, without any preconceived notions, and have tried not to become hyperenthusiastic.

Just as the occasional patient may fail to receive symptomatic relief after an appendectomy, so there may be patients who do not obtain symptomatic relief, or are apparently worse, after resection. Close analysis of this group will show that the obstruction has not been completely relieved in some cases; in others, the complaints are due to an overlooked carcinoma or the presence of a diverticulum or, in a rare instance, are due to a large hydronephrosis.

Regarding the number of resections: In my experience it has been necessary in about 10 per cent of the cases to perform a second resection, and, as I mentioned in the paper, the cases in whom a second resection was necessary fall into three groups: first, the cases of very large hypertrophies in whom it is evident that a second resection was necessary to remove the obstruction; second, the cases of carcinoma that recur or that continue to grow and produce obstruction; third, the small number of cases in which not enough tissue had been taken off at the time of resection. There were five patients who required three resections each.

I agree with the statement that has been made that it is unfortunate that there has been so much sales promotion regarding various types of machine and not enough stress has been placed on thorough grounding in pathology and endoscopy of the prostatic urethra and bladder neck.

Regarding the question of incontinence, I would state that we have no patients with complete incontinence; there were two patients who had some slight trouble and they are rapidly improving.

I wish to emphasize what Doctor Lower said, namely, that this is not a simple operation and that technically it is very difficult to carry out.

Regarding the selection of cases, I have tried to emphasize in this paper that this is still a moot point for discussion. An interesting thing was noted in this series of cases, namely, that the patients who had late secondary bleeding, that is, who had bleeding two or more years after resection, were all patients who had very large hypertrophies in the beginning.

I wish to agree with the rest of the points made by Doctor Cabot and Doctor Lower and it seems to me that this procedure will take its proper place in our armamentarium in the very near future.

OBSTRUCTIONS ABOUT THE MESENTERY IN INFANTS

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REPEATED vomiting in infancy is always a cause of great concern to the parents and to the attending physician. When the baby cannot retain his feedings in the first weeks of life the usual procedure is to try various modifications in the diet. If after several failures, and the response even to thickened feedings and atropine therapy is without result, an organic obstruction of some type is likely to be found the underlying cause. The most common etiologic factor of organic obstruction in infancy is hypertrophic pyloric stenosis. But there are a certain number of obstructions which make their presence known almost as soon as the baby takes anything by mouth. These obstructions can be differentiated from hypertrophic pyloric stenosis. They occur in or about the duodenum and are usually in close anatomic relationship to the mesentery of the small intestine. We wish to consider this group of obstructions in this communication. The group includes the duodenal atresias; the internal herniae; and the anomalies due to faults in migration, descent and fixation acting on the first portions of the small intestine.

The earliest recorded case of congenital intestinal occlusion is credited to Calder who, in 1733, according to Cordes[1]* described an atresia of the duodenum. In 1901, Cordes collected from the literature only 56 cases of duodenal atresia and stenosis. Two years later Kuliga[2] made an extensive study of congenital occlusions of the entire intestinal tract based on 189 reported cases. In 1922, Davis and Poynter[3] increased this number to 392. In other words, the reported cases averaged two per year over a period of approximately 200 years, which gives a false impression of the frequency with which the condition occurs. While congenital intestinal anomalies leading to complete and partial obstruction are unusual, they are not rare. Ladd,[4, 5] who reported 60 cases from the Children's Hospital of Boston, states that the fact that he has "encountered ten cases in a comparatively short time makes me feel that they (congenital anomalies) have been overlooked in the past." He feels that the "frequency of postmortem reports of the cases and the infrequency of these conditions in adult life indicate a high mortality without surgical intervention."

Since Cordes' contribution other reports stressing various phases of congenital occlusions have appeared. Among these should be mentioned those of Pehu and Auberge,[6] Thorndike,[13] DeSanctis and Craig[7] and most re-

* 1752 according to Davis and Poynter.[3]

cently Ladd. Spriggs[8] gives an excellent and detailed description of the microscopic findings in cases of stenoses, as do Davis and Poynter. The latter feel that the fundamental etiologic factor is primary sclerosis of the regional vessels, the changes in the bowel wall being secondary to the resulting circulatory disturbances.

Congenital intestinal occlusions are commonly classified as intrinsic and extrinsic. The pathology of the atresias varies from a complete absence of a portion or portions of the intestine to all grades of intestinal fibrosis and diaphragms occluding the lumen. Atresias occur more commonly in the duodenum than in any other part of the gastro-intestinal tract, 30 per cent according to von Koos[9] and Davis and Poynter. The extrinsic occlusions form an interesting group and it may be said that in general they result from faulty or incomplete intestinal rotation and hyperperitonealization during fetal development. Internal herniae as a cause of obstruction are encountered less frequently than short mesenteric arteries, incomplete intestinal rotation, abnormal peritoneal bands and adhesions.

An understanding of the embryologic development with the changes in anatomic relationships of the gastro-intestinal tract will help the surgeon to untangle these complicated anomalies which he encounters at operation. A fairly accurate picture of the process has been pieced together for us by studies on human embryos and from observations in comparative anatomy.[18] It is presented as a theoretical motion picture although the evidence is made up from a series of still exposures often with great unfilled gaps between. When one realizes the many opportunities that there are for something to upset the orderly progression it is amazing that these anomalies are so relatively uncommon.

The stomach moves down from the cervical into the lower thoracic region during the sixth and seventh week of embryonic life. It hangs vertically with a right and a left surface until a more rapid growth of the dorsal or posterior border and its mesentery takes place. This inequality in growth rate leads to the development of a relatively freely movable greater curvature and a relatively fixed lesser curvature. The fundus also evolves as an outpouching on this dorsal border. The resulting disproportion between the anterior and posterior border of the stomach leads to a rotation, the greater curvature moving towards the left. The former right surface finally becomes posterior and the left surface anterior in position. During this same time the duodenum is giving rise to the buds which develop into the liver, biliary passages, and the pancreas. The duodenum then becomes occluded by proliferation of its lining epithelium. It remains in this condition so that the lumen is blocked until the sixth week. Then, normally, the epithelium is absorbed again. In rare instances this does not occur but the epithelium becomes organized. This mechanism results in the various types of atresia encountered in the duodenum as well as elsewhere in the jejunum and ileum. These atresias may be single or multiple, partial or complete. The occlusion may be a solid cord of some extent or a simple membrane. Cases 1 and 2

illustrate this type of anomaly. Atresia in the duodenum frequently occurs just above the opening of the common duct, but we have not met such a case in our series.

The mobile duodenum undergoes a rotation and fixation of its own from the sixth to the ninth week. It is forced towards the right side of the abdomen by the rotation of the stomach, and there twists on its axis. This causes an alteration in the position of the common duct and the pancreatic outgrowths so that they occupy more nearly their adult positions. The duodenum then becomes fixed to the region of the gallbladder and liver, fused with the dorsal wall, and suspended behind the root of the mesentery by a fibromuscular band, Treitz's ligament. The superior mesenteric artery thus passes in front of the terminal part of the duodenum.

That portion of the intestinal tract supplied by the superior mesenteric artery—the midgut—starts out as a U loop within the umbilical cord. In the sixth week of life the cecal bud becomes apparent. By the ninth week coils of lower jejunum and ileum have formed in the cord and an upper jejunal coil distal to the duodenojejunal attachment. The loops in the cord are drawn into the abdominal cavity by retraction bands in the mesentery during the tenth week. The operation of rotation of the bowel which is necessary before its fixation takes place appears to be greatly helped by its shape and position in this special umbilical sac. The portion of the midgut from the duodenum to the vitelline artery passes in behind the artery from right to left. The portion of the midgut from the vitelline artery to the midtransverse colon passes in front of the artery from left to right. By the third month the U shaped loop has become twisted around the axis of the superior mesenteric artery.

The hindgut is drawn up and molded into place by pressure of the small bowel against it. It usually fuses with the posterior wall assuring its fixation. Towards the end of the third month the large bowel occupies an oblique position across the abdomen from the right iliac crest to the left hypochondrium. The rotation has brought the mesentery of the cecum and ascending colon into contact with the duodenum. The cecum thus comes to rest in front of the lower part of the right kidney near the gallbladder in the majority of fetuses. It remains there until about the time of birth. At the time of birth the ascending colon moves toward the right iliac fossa, elongating in the process—descent. The liver and the gallbladder move upwards providing more room for fixation of the ascending colon at the hepatic flexure. The adhesions which the ascending colon forms in its fixation, just before and just after birth as the cecum descends to its final position, offer many opportunities to produce anomalies.

If there are unequal tension pulls on the retroperitoneal duodenum it may be angulated or twisted on itself, especially since it may be caught between two opposing forces—those causing its firm anchorage at Treitz's ligament and those pulling towards the right iliac fossa. Under these conditions it is not strange that we have encountered such partial obstructions as are recorded in Cases 5, 6, 7, 8, and 9. These result from hyperfixation

of the peritoneum about the duodenum giving partial obstructions from plications or torsions of its lumen. In Cases 10 and 11 we have examples of obstructions due to failure in descent of the cecum. These patients presented an exact duplication of the position and attachments of the great bowel at the end of the third month of development. But their close attachments prevented the duodenum from functioning properly, especially in Case 11 where a well formed cystocolic membrane was exposed. In Case 4 we have a most interesting example of one of these developmental anomalies. It is classed as a retroperitoneal hernia by most surgeons. We believe that it represents an abnormal fixation of the mesentery. When the rotation occurs in a direction opposite to the normal, the duodenum and mesentery come to lie in front of the transverse colon instead of behind it. These cases are a great puzzle to the surgeon unless he can expose the root of the mesentery. Then, the whole bowel unrolls rapidly. At the finish of such an operation all the small bowel will occupy the right and all the large bowel the left side of the abdominal cavity. This is the condition as it is in development before any rotation has taken place.

When there is failure of complete fusion at the duodenojejunal junction, a deep fossa may be left into which jejunum may herniate. Case 3 is an example of a very unusual herniation into the lesser peritoneal cavity through such an opening.

Herniation of the small intestine into the lesser peritoneal cavity is uncommon. Moynihan,[10] in his monograph on retroperitoneal hernia, cites only four cases. The bowel gains access to the lesser peritoneal cavity by two routes, the more common being through the foramen of Winslow. Dewis and Miller[11] reported 34 cases. Less frequently as in the present case, herniation occurs through the transverse mesocolon. Heaney and Simpson[12] reported 21 cases of this type. The herniated intestine may remain in the lesser peritoneal cavity or it may partially emerge by forcing itself through the gastrohepatic or gastrocolic omentum to again occupy, more or less, its normal position in the abdominal cavity. Occasionally herniae through the transverse mesocolon emerge through the foramen of Winslow.

In reported cases increased intra-abdominal pressure due to vomiting caused by associated lesions (ulcer or carcinoma of duodenum and stomach) was felt to be responsible for producing a tear in the transverse mesocolon. Its occurrence in adult life makes it probable that in most instances it is an acquired lesion. There seems to be little question that in the present case the defect in the mesocolon was congenital.

CASE REPORTS

Case 1.—No. 34798. E. S., a white female infant, the fourth in the family, was born in the Strong Memorial Hospital, May 1, 1930. The father and mother were healthy with negative Wassermanns. The pregnancy had been normal, the delivery spontaneous, and at birth the child was apparently normal weighing 2,600 Gm. (5.7 lbs.). The patient was seen in consultation by the pediatricians on May 3 because of vomiting and failure to pass urine and meconium. Vomiting started 48 hours after birth. Regurgitation

was frequent, occurred immediately after feeding and on some occasions consisted of a thin brownish fluid containing "coffee ground" material.

Physical examination showed a dehydrated female who appeared normal. Abnormal findings were limited to the abdomen which was thin walled and distended in the epigastrium but scaphoid in the lower abdomen. A sausage shaped mass extended diagonally across the epigastrium from the left costal margin to the right flank about on a level with the umbilicus. Across this mass peristaltic waves were visible, passing usually from left to right but occasionally from right to left. The mass was soft and doughy. The liver edge was palpable 1 cm. below the costal margin. No other masses were palpable. Bladder dulness was not increased to percussion.

Frequently repeated subcutaneous saline infusions were given. A catheter introduced into the rectum passed readily without encountering an obstruction. A small amount of normal saline which was introduced was readily expelled and with it a thick tenacious plug or cord of meconium 10 cm. in length. Following this more meconium was passed. The size of the abdominal mass remained unchanged. On May 4, the third day, the signs and symptoms were unchanged. A barium enema showed the colon patent and filled up to the hepatic flexure. At this point there was a "redundant loop of bowel distended with gas." The barium did not fill the ascending colon or cecum. The patient was first seen by the surgeons May 5, the fourth day, at which time her condition was grave. She was almost moribund. Her weight was 2,260 Gm. (4.95 lbs.), a loss of 340 Gm. (.74 lbs.). The temperature was 35° C. The abdominal signs were unchanged.

Operation was performed immediately under ether anesthesia, a right rectus incision being made. The stomach and duodenum were tremendously distended, the diameter of the latter being about 8 cm. and its walls of tissue paper thinness. The duodenum was bound down by abnormal peritoneal bands which were released. The transverse colon and stomach were reflected upward and Treitz's ligament identified. The jejunum appeared normal though small, being about the size of an ordinary lead pencil. Dilatation of the duodenum was found to extend to the third portion at the point where the superior mesenteric vessels crossed, these having been reflected upward and any possible constricting effect there relieved. Gentle pressure was exerted on the dilated duodenum but no gas passed beyond the point of obstruction. It was thus apparent that the obstruction was intrinsic in the retroperitoneal portion of the duodenum and due either to an occluding membrane or an atresia. Relief of the obstruction depended upon a short circuiting anastomosis. Duodenojejunostomy, the logical procedure, was considered inadvisable and almost impossible due to the thinness and delicacy of the duodenal wall as a result of distention. It was questionable if the thinned out walls would hold the sutures. A posterior gastro-enterostomy was performed without clamps, using silk sutures throughout. The stoma was approximately 4 to 5 cm. The patient's condition at the end of the operation was poor.

The immediate postoperative recovery was excellent. The temperature rose to 39° C. the day after operation, returning to normal on the third day. Subcutaneous saline infusions were given regularly. Feedings were started the day after operation. The patient vomited once and thereafter feedings were given by gavage; there was no further vomiting. She took the bottle on the third day after operation and from then on progressed satisfactorily. She regained all weight lost and when discharged 15 days after operation weighed 2,620 Gm. (5.7 lbs.). A normal stool was passed on the second day after operation. Urinary function became normal. A gastro-intestinal series two days before discharge showed a dilated stomach and a well functioning gastro-enterostomy (Fig. 1).

Unfortunately, it was soon recognized that the patient was a Mongolian idiot. Her course from the gastro-intestinal standpoint was entirely satisfactory for one year. Irregular attacks of vomiting and diarrhea occurred about a year after operation. These

FIG. 1.—Case 1. Roentgenogram showing a dilated atonic stomach and duodenum. Taken after gastro-enterostomy.

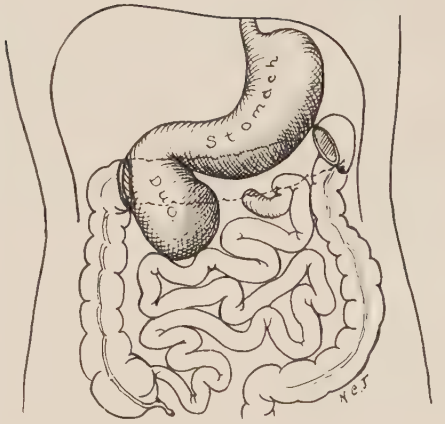

FIG. 1*a*.—Case 1. Atresia of duodenum. Condition found at operation. Absence of third part of duodenum. Confirmed at autopsy two years later.

persisted for a period of one or two months and then ceased. In January, 1932, 20 months after operation, she was readmitted to the hospital with a diagnosis of pneumonia, to which she succumbed.

Postmortem examination showed bilateral bronchopneumonia, acute bronchitis, bilateral otitis media, acute splenic tumor, atresia of the duodenum with complete absence of the third portion, a healed, patent gastro-enterostomy and a patent foramen ovale.

Case 2.—No. 61269. J. L., an apparently normal female, the fourth child, born in the Strong Memorial Hospital, May 22, 1932, began vomiting on the fifth day. Changes in formula, antispasmodics, *etc.*, failed to relieve symptoms. During the first 11 days of life the weight decreased from 3,350 Gm. (7.37 lbs.) to 2,620 Gm. (5.76 lbs.). Physical examination on June 3 was essentially negative except for visible peristaltic waves just below costal margins, traveling from left to right, and incomplete closure of the umbilicus. A small amount of barium administered the same day showed dilatation of the first portion of the duodenum with complete obstruction about the beginning of its second part. Adequate fluid intake had been maintained by administration of subcutaneous saline.

Operation was performed on June 4. A tremendously dilated duodenum presented itself. The transverse colon was small and occupied its normal position. It was reflected from its normal attachment allowing exposure of the retroperitoneal portion of the duodenum which was explored as far as Treitz's ligament. It was seen that a considerable portion of the ascending colon was also retroperitoneal and it was released from its retroperitoneal position. A clear view of the entire small intestine was thus possible and no obstruction was apparent at any point except where the duodenum entered its retroperitoneal position. The retroperitoneal duodenum was small and collapsed but appeared normal. The pancreas appeared normal. There were several small aberrant pancreatic nodules in the wall of the dilated duodenum. Pressure on the dilated duodenum failed to force any gas into the distal retroperitoneal duodenum. The duodenum was opened longitudinally at the point of obstruction exposing a diaphragm about 2 Mm. in thickness apparently completely blocking the lumen of the bowel. Bowel contents were carefully aspirated. A normal bowel lumen was readily demonstrable below the diaphragm. The diaphragm was completely removed by monopolar desiccation, which adequately controlled all hemorrhage. The intestinal lumen was restored by suturing the bowel transversely with silk and reenforcing with mattress sutures of the same material (Fig. 2).

While at no time during recovery was the patient's condition alarming, considerable difficulty was encountered. A 50 cc. transfusion was given following operation and numerous subcutaneous saline infusions during the first four days. Vomiting and distention were troublesome until the twelfth day postoperative. What feedings were retained were supplemented by saline infusions. Gastric lavage was used frequently. It was not until about two weeks after operation that the patient stopped losing weight and began to retain feedings with any regularity. Fluoroscopy showed slight delay in emptying of the stomach but free passage of barium along the upper intestinal tract. All vomiting finally ceased and the patient was discharged July 13, 1932, on the thirty-ninth postoperative day. The weight on discharge was 3,440 Gm. (7.48 lbs.).

The patient was readmitted on two or three occasions during the next few months for coryza, otitis media and on one occasion because of diarrhea and vomiting, neither of which was observed during a subsequent five day stay in the hospital. The general course has been one of constant improvement and normal development. A gastro-intestinal series July 6, 1932, a month after operation, showed some enlargement of the stomach and dilatation of the duodenum but no obstruction to the flow of barium. In December, 1932, at age of six months, she was observed in the hospital for a period of six days. She was admitted because of apparent abdominal pain and constipation. Thorough study showed no evidence of obstruction. In October, 1933, a year and five months after operation, she was readmitted because of intermittent vomiting over a

FIG. 2.—Case 2. Dilated atonic stomach and duodenum as shown by roentgenogram.

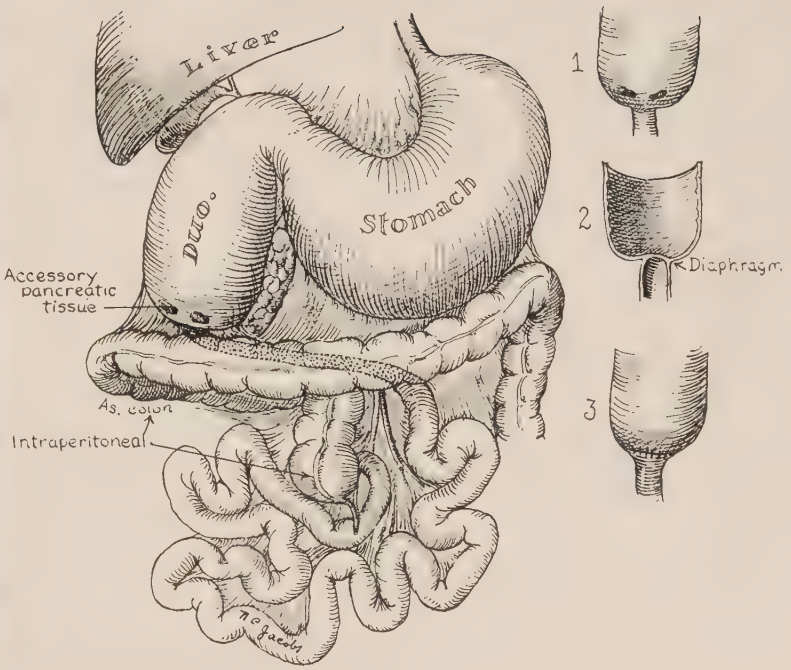

FIG. 2a.—Case 2. Atresia due to diaphragm. Condition found at operation. Repair as carried out to right indicated in Inserts 1, 2, and 3.

period of two months. A gastro-intestinal series at this time showed no definite obstruction. The stomach emptied itself slowly and passage of the barium through the intestinal tract was slow but no point of obstruction was noted. During 1934 there were no gastro-intestinal complaints. Illnesses during 1934 consisted of pyuria on two occasions and whooping cough, otitis media again in 1935, also measles and furunculosis.

Case 3.—No. 35271. E. W., white, male, the third child in the family, age three days, was referred to the Strong Memorial Hospital May 23, 1930, because of persistent vomiting since birth. The antecedent history was entirely negative, both parents being normal adults. The pregnancy and delivery had been uncomplicated. At birth the patient was apparently a normal healthy infant weighing nine pounds. Breast feedings had been started immediately. Vomiting, projectile in type, started immediately after the first feeding and had persisted until the time of admission to the hospital. It occurred regularly about 15 minutes after each feeding and apparently the entire feeding was vomited. The stools, at first greenish in color, became slightly yellow on the third day. There had been a slight discharge from the left eye since birth. On admission to the hospital the patient weighed seven pounds, 14 ounces and physical examination was entirely negative except for a slight conjunctivitis of the left eye. Examination of the blood presented no abnormalities in the cellular elements but the chemical analysis disclosed a non-protein nitrogen of 74 mg. per 100 cc., while the chlorides were 675 mg. per 100 cc. Stool examination was negative. Vomiting persisting despite treatment, a small amount of barium was given by tube the day after admission and its course followed by fluoroscopic examination. The esophagus, stomach and duodenal cap appeared normal except that the latter remained filled and very little if any of the barium progressed into the distal portions of the duodenum. At the end of an hour practically all the barium was still in the stomach, only traces being discernible in the jejunum, which showed vigorous peristalsis. The course of the barium was observed at intervals and at the end of 19 hours no barium remained in the small bowel, about one-quarter being in the ascending colon and the remainder being still in the stomach. It was quite apparent that the infant was suffering from a partial obstruction of the upper gastro-intestinal tract distal to the first portion of the duodenum, the exact cause being undetermined (Fig. 3). With the hope that the underlying cause was such that the obstruction might eventually relieve itself, operative treatment was delayed. During this observation period the infant was given various formulae without any improvement. Dehydration was combated by repeated subcutaneous injection of normal saline, 1,200 cc. administered over a three day period.

Operation was performed May 27 under ether anesthesia. On opening the abdominal cavity through a high right rectus incision, the duodenum, somewhat dilated, was found to occupy a high position. The stomach and pylorus appeared normal. Above the lesser curvature of the stomach in the lesser peritoneal cavity could be seen dilated loops of small bowel. The transverse colon and stomach were reflected upward and Treitz's ligament identified. The jejunum was carefully picked up and an attempt made by gentle traction to deliver it into the wound. There was a slight resistance which on being released, disclosed, in the region of Treitz's ligament, a dilated segment of jejunum which immediately exhibited vigorous peristalsis. At each end of this dilated portion of jejunum there were apparent, across the bowel, definite markings, indicative of constriction. The release of the obstructed segment and its appearance in the wound occurred so rapidly that the opening by which it gained access to the lesser peritoneal cavity was not identified. The intestinal loops had disappeared from the lesser peritoneal cavity and as a rapid examination of the remainder of the gastro-intestinal tract showed no other abnormality, the abdomen was closed.

The postoperative convalescence was entirely satisfactory. There was a moderate amount of vomiting during the first 48 hours during which time an adequate fluid intake was maintained by subcutaneous administration of normal saline solution. The subsequent course in the hospital was uneventful, the infant retaining the prescribed formula

without difficulty. At the time of discharge from the hospital June 3 the infant weighed 3,480 Gm. (7.65 lbs.). About two weeks after discharge there occurred a mild gastro-intestinal upset which was readily controlled by a change of formula. On June 25, the patient weighed 4,450 Gm. (9.79 lbs.).

Case 4.—No. 56990. T. C., a white male, age two and one-half months, the sixth child, was admitted to the Strong Memorial Hospital January 29, 1932, because of convulsions. At birth the patient had appeared normal and weighed nine and one-quarter pounds. Projectile vomiting began immediately after birth and still persisted at the time of admission. It occurred usually soon after feeding. At times it would cease for as long as three to four days. Sometimes evening feedings were retained and vomiting would occur in the morning, the vomitus under such circumstances consisting

FIG. 3.—Case 3. The peculiar shadow noted along the lesser curvature is due to a loop of jejunum herniated into the lesser peritoneal cavity. The faint shadow back of the stomach indicates the position of this loop of bowel from its entrance in the para-duodenal fossa.

of the feeding of the previous evening. Occasionally it was yellow or green but generally it consisted of the food taken at previous feeding. The patient was breast fed for two weeks, following which various formulae were tried. Constipation was marked until shortly before admission. Three weeks before admission, while feeding, the patient suddenly became unconscious, remaining so for five to ten minutes. The evening before admission he had the first convulsion, which was repeated several times during the night. Following the first convulsion abdominal distention developed. This was relieved by an enema. There had been slight jaundice for the first two weeks of life.

Physical examination showed a well developed but undernourished male infant, lying with eyes open and eyeballs rolled upward and rotated to the right. The extremities were flexed in a clonic state, but could be extended. Fists were clenched, no cyanosis. While palpating the abdomen, the patient suddenly relaxed and the eyes straightened.

Respirations were irregular. Suddenly the left arm began to jerk, the hand was again clenched and the patient became unconscious. Respirations stopped, the heart beat which had been forceful became distant. Artificial respiration stimulated breathing but respirations continued to be shallow. Convulsions continued throughout the examination. The heart and lungs were normal. The abdomen was moderately distended with the liver edge palpable at the costal margin. The tendon reflexes could not be elicited. There was no clonus. Chostek and Babinski were negative. The temperature was normal.

Laboratory examinations showed: red blood cells, 3,600,000; hemoglobin, 60 per cent; white blood cells, 15,200. The platelets were slightly decreased. The differential was normal. The urine and stool were negative. Blood chemistry showed calcium 9.6 mg., phosphate 5.6 mg., chlorides 410 mg., CO_2 70 vols. per cent. The Wassermann was negative. Lumbar puncture showed a pressure of 15, clear fluid, Pandy negative and one cell.

During the first few days in the hospital the patient's temperature varied from normal to 39.5° C. Vomiting persisted with more or less regularity and was projectile in type. Examination of the vomitus failed to reveal any occult blood. The stools were watery. Five days after admission a left myringotomy yielded a moderate amount of pus. The fluid level was maintained by subcutaneous saline as indicated. Eight days after admission a transfusion was given. On February 16, 1932, a roentgenologic gastro-intestinal study showed some pylorospasm, but more important, a marked delay in emptying of the duodenal cap due to some obstruction in the duodenum just beyond the cap. The duodenum was found to be in an abnormal position. All possible variations of formulae and antispasmodics were tried in spite of which vomiting persisted, and even increased in severity and frequency. Despite the patient's poor condition from infection (ears), *etc.*, laparotomy was performed February 26.

Operation was started under local infiltration which was later supplemented with light ether. The stomach and duodenum were distended. Fine filmy peritoneal bands crossed the duodenum in several places. The omentum was attached to the duodenum. The common duct and pancreas were easily exposed and visualized. The duodenum was dilated to the point where the mesenteric artery and vein crossed. The latter were incorporated in a constricting band. There was no apparent exit of small bowel from this point. Loops of small bowel were visible retroperitoneally in the left side of the abdomen. Involved in the above mentioned constricting band were three loops of large bowel, part of which were adherent to the duodenum in a retroperitoneal position. The cecum and appendix, crossing on top of these loops, were securely fixed by additional bands (Fig. 4). Gentle traction on exposed large and small intestinal loops failed to release this complicated tangle in any way. The lateral attachment of exposed bowel was divided far over on the right side and down to the posterior abdominal wall. It then became apparent that the ascending colon and cecum had looped back around this lateral attachment. The transverse colon had become attached by a peritoneal band in front of the duodenum. By freeing the cecum and appendix it was possible to draw out from its retroperitoneal sac the entire small intestine which was collapsed and dark blue in color from obstruction of its circulation. By careful dissection down to the mesenteric vessels the transverse colon was freed, after which it was possible to untwist the small bowel, thus entirely relieving the obstruction. With the untwisting of the small bowel it immediately flushed to a bright pink color, indicating the release of the obstruction to the blood supply. The small bowel now occupied the entire right side of the abdomen and entered directly into the large bowel, which occupied the entire left side and was not attached in any way to the posterior abdominal wall. The whole gastro-intestinal tract was thus one continuous tube from the pylorus to the sigmoid. The opening in the small mesentery was closed with silk. The operation was surprisingly well tolerated.

The postoperative recovery was entirely satisfactory. Except for some vomiting

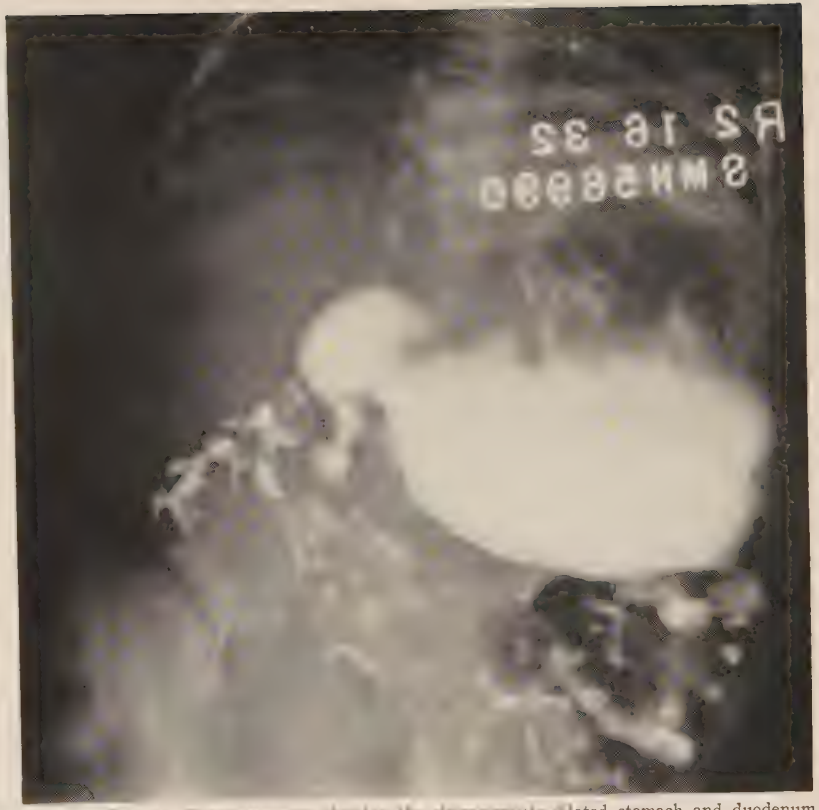

Fig. 4.—Case 4. Roentgenogram showing the characteristic dilated stomach and duodenum.

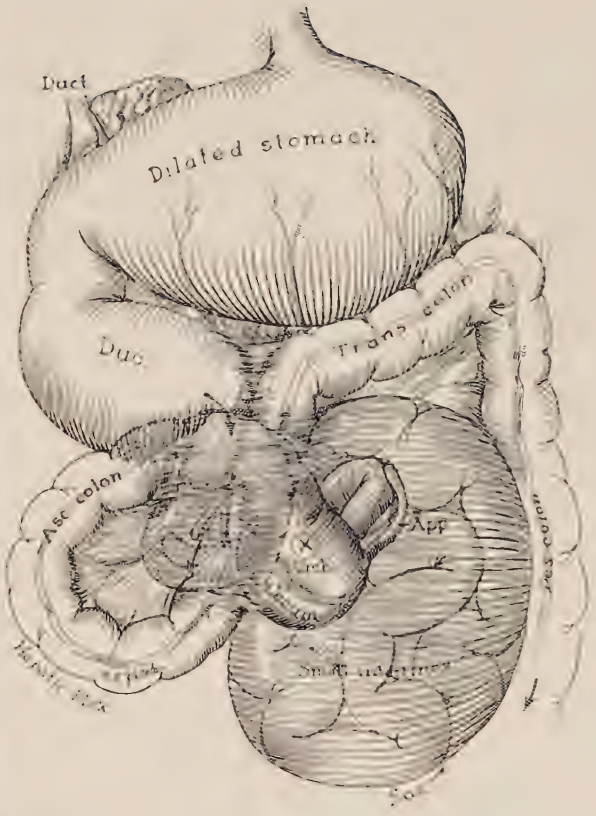

Fig. 4a.—Case 4. The condition found at operation. This is not an internal hernia but a reversed rotation of the midgut on the mesentery. This obstruction is easily handled by exposing the root of the mesentery and twisting the bowel in the reverse direction.

immediately following operation, feedings were well taken and retained. There was a slow steady gain in weight.

Case 5.—No. 107422. J. G., the fifth child in the family, was born prematurely in the Strong Memorial Hospital, September 26, 1935. She weighed 2,480 Gm. (5.45 lbs.) at birth. Vomiting (regurgitation) began on the third day and became projectile in type on the seventh day. It occurred immediately after feeding and consisted of the food previously ingested. Thrush which developed on the sixth day was successfully treated. No bowel movement from October 2 to October 7. An enema on October 7 effected the discharge of some greenish-brown fecal material. There was subsequently a normal stool which was guaiac positive. Changes in formula, antispasmodics, refeeding and gavage failed to control symptoms and the patient continued to lose weight. On October 7, 1935, at the age of 12 days, the weight was 2,300 Gm. (5.0 lbs.). Frequent saline infusions had been given.

Physical examination at this time showed a small, emaciated, premature infant with pinched facies. There was slight cyanosis of lips. The head appeared large in proportion to the size of the body and the anterior fontanelle was soft. There were white membranous patches on the inside of each cheek and on the tongue. The umbilicus was practically healed and there was slight herniation. Otherwise the abdomen was negative.

A gastro-intestinal series made on October 9 showed the stomach and upper intestinal loops filled with gas. The barium left the stomach very slowly.

Operation was performed October 11 under ether anesthesia through an upper right rectus incision. The stomach was large and dilated. The pylorus was normal. The duodenum was dilated and was attached throughout its entire length by peritoneal bands which ran across the duodenum and transverse colon, anchoring it in several kinks making an accordion pleated type of obstruction. The attachment of the transverse and ascending colon was freed to the cecum, after which the large bowel was rotated to the left and the adhesions between the duodenum and mesentery were carefully divided all the way to Treitz's ligament. This allowed the kinked portion of the duodenum to straighten out and assume a normal position. The abdomen was closed with silk.

The postoperative course was satisfactory although somewhat difficult. One transfusion was given postoperatively. Subcutaneous administration of saline and glucose was given repeatedly and regularly during the first five days following operation. Vomiting occurred irregularly during the first ten days, but there was none thereafter. Her general condition improved steadily and at the time of discharge on November 27, aged 62 days, her weight was 3,340 Gm. (7.3 lbs.).

The child was last seen March 2, 1936, aged five months, at which time she was symptom free and weighed 5,840 Gm. (12.8 lbs.), an increase of 3,440 Gm. (7.4 lbs.) since birth.

Case 6.—No. 105930. J. B., a white, female, first child, age 34 days, was admitted to Strong Memorial Hospital September 20, 1935, because of persistent vomiting. Vomiting began about the ninth or tenth day of life and at first occurred only once daily or on alternate days. It always occurred after feedings and was projectile in type. Symptoms increased in severity and frequency up to the time of admission despite all possible variations in formulae and antispasmodics. Refeeding was instituted successfully, the patient never having vomited after this procedure. Shortly before admission peristaltic waves were noted in the epigastrium, passing from left to right. The weight had increased 970 Gm. An interesting point in the family history is that one year before the birth of the patient, the mother had had an exploratory laparotomy, at which time congenital bands causing a partial intestinal obstruction were released.

Physical examination was essentially negative. The only abnormalities noted were areas of capillary hemangiomata over the face and trunk and a somewhat protuberant

abdomen showing peristaltic waves in the epigastrium, coursing from left to right. There was some anemia; red blood cells, 3,160,000; hemoglobin, 11.0 Gm.

Operation was performed September 21, 1935, under ether anesthesia. The stomach and pylorus were normal. The duodenum was dilated two to three times its normal size and was tightly bound down by a series of peritoneal adhesions. These were freed, allowing the duodenum to assume its normal position. The abdomen was closed with silk.

The patient tolerated the operative procedure well. She was given a transfusion and frequent subcutaneous saline infusions following operation. Feedings were given by gavage, the tube being left in place due to the presence of some distension, which soon subsided. There were two normal stools on the second postoperative day. The patient was discharged on the third postoperative day. Her subsequent course has been entirely satisfactory and without symptoms.

Case 7.—No. 97611. N. B., a white male infant, age two days, was admitted to the Strong Memorial Hospital December 21, 1934, for repair of cleft palate and hare lip. Physical examination was negative except for the local condition, *i.e.,* complete left unilateral hare lip and a complete bilateral cleft palate.

The patient vomited every feeding following admission. Stools consisted mostly of meconium with a small amount of formed stool. Changes in formulae and antispasmodics were without avail. Five per cent glucose was given subcutaneously several times each day. Four days after admission, on December 25, a small amount of barium was given. Fluoroscopy showed marked gastric retention with only a minute amount of barium passing through the pylorus. Most of the barium was still present in the stomach after three hours. There was no dilatation of the duodenum. Conservative treatment was continued without any improvement until December 28, when operation was performed.

Operation was started under local infiltration, but it was necessary to change to drop ether in order to adequately explore the abdominal cavity. The stomach, pylorus, and duodenum were normal. The colon was reflected upward and Treitz's ligament identified. Just beyond Treitz's ligament was a marked twisting of the upper jejunum due to hyperperitonealized bands. Salt solution injected into the stomach flowed readily through the pylorus and the duodenum but stopped at Treitz's ligament. The peritoneal bands were divided, releasing the intestinal kinking. There still seemed to be an obstruction at one point. A membrane obstructing the lumen was suspected and the bowel was opened longitudinally. No intrinsic obstruction was found and the opening was closed by suturing transversely. The injected saline then flowed readily beyond this point.

The patient continued to vomit for three days following operation during which time numerous glucose and saline infusions were given. Beginning on the fourth day, feedings were well taken and retained except for an occasional attack of vomiting. Feeding of course was difficult due to the cleft palate. However, the patient's condition improved steadily so that by January 15 the cleft premaxilla was approximated by silver wire under ether anesthesia. The patient expired suddenly at 5 P.M. the day of operation.

Postmortem examination, besides a large frothy bloody mucous mass filling the posterior pharynx, showed focal areas of bronchopneumonia, acute bronchitis, subpleural ecchymoses, and hemangiomata of the tricuspid and mitral valves.

Case 8.—No. 83494. B. C., white, female, age ten months, was first admitted to the Strong Memorial Hospital, December 20, 1933, at the age of four months because of persistent vomiting since birth. The vomiting occurred as often as five to six times per day, three to four hours after feeding, and was not projectile. The vomitus consisted of curdled, partially digested milk, and did not contain bile. The amount varied considerably but for the most part represented only a small amount of the previous feeding taken. Stools had been normal. Physical examination was entirely negative. The blood and urine were normal. Fluoroscopy of the intestinal tract after barium showed a large atonic stomach with marked delay in emptying. She was readmitted

FIG. 5.—Case 8. Roentgenogram showing passage of barium into small intestine, but delay in an egg-shaped loop seen through the gas filled stomach.

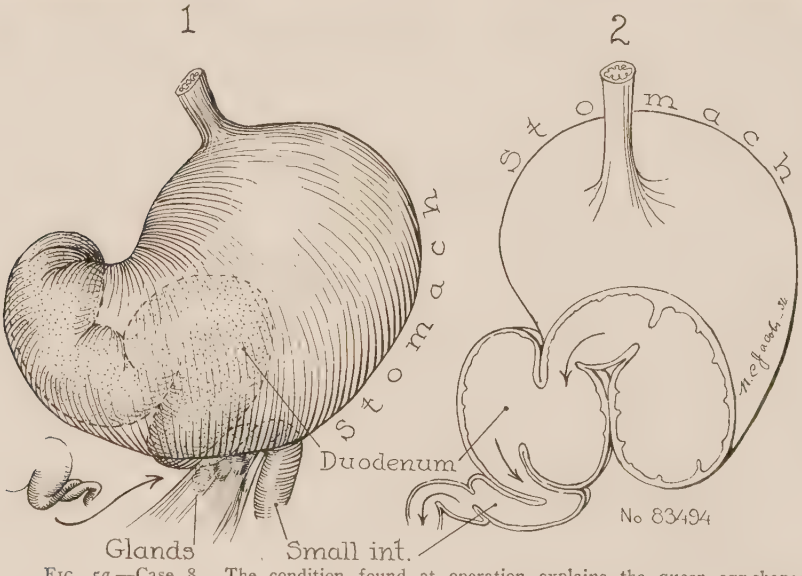

FIG. 5a.—Case 8. The condition found at operation explains the queer egg-shaped shadow in the roentgenogram. This is a torsion and plication obstruction freed by releasing the bands.

for the same symptoms on June 11, 1934, having been carried along on a medical regimen during this time. Vomiting was less frequent. On one occasion peristaltic waves were seen. However, for a week or two prior to admission vomiting increased and the patient lost one pound in one week. In the interim, symptoms had changed somewhat. Vomiting frequently occurred immediately after feeding, and at times was projectile in type. Bile had been present in the vomitus on two occasions. There had been increasing constipation.

Physical examination showed no abnormalities. Abdominal examination was negative. The blood and urine were negative. Examination of the gastro-intestinal tract after the injection of barium showed no dilatation of the stomach but considerable delay in emptying, a fairly large amount being present after six hours (Fig. 5).

Operation was performed June 13, 1934, under ether anesthesia, a right rectus incision being made. The stomach and first portion of the duodenum were dilated. The transverse and ascending colon were freed from the lateral abdominal wall and rotated to the left, giving a good exposure of the retroperitoneal duodenum. In the third portion of the duodenum behind the mesentery there was a longitudinal torsion of the intestine in two places, caused by hyperfixation. The duodenal attachments were freed in these places allowing the duodenum to straighten out. Gas promptly passed into the first portion of the jejunum. The abdomen was accordingly closed (Fig. 5).

The postoperative course was satisfactory though somewhat difficult. Subcutaneous saline infusions were given as indicated. Feeding was resumed the evening of the day of operation. Vomiting occurred at irregular intervals, but never was persistent or alarming. The patient was discharged from the hospital 15 days after operation in excellent condition.

The patient was last seen October 23, 1935, one year and four months after operation. Her condition was excellent and development had been normal. The only vomiting spell noted since the first two weeks after discharge was associated with an acute upper respiratory infection.

Case 9.—No. 89644. R. S., a baby boy, was born in the Strong Memorial Hospital May 21, 1934. He appeared normal. The birth weight was 6.9 lbs. In the first few days of life he vomited mucus on several occasions. Upon nursing at the breast he regurgitated small amounts after each feeding. On the fifth day he vomited once forcefully. The stools were entirely meconium for the first three days and there were no stools for the next two days. The weight dropped till on the fifth day it was 5.4 lbs. He became jaundiced and dehydrated. General examination was negative. The physicians thought that he might have pyloric stenosis and dehydration jaundice. He was given repeated saline infusions. Several different formulae were tried. Atropine 1:1000 was used, one drop before each feeding. In spite of this the vomiting continued and on one occasion it contained a small amount of blood. He was given one teaspoonful of barium in two ounces of his formula and observed under the fluoroscope. The mixture seemed to be delayed at the cardia for some time and was then regurgitated. An esophagoscope was accordingly passed through the cardia and no obstruction was encountered. The pediatricians then placed the child on a thickened formula, each feeding being preceded by gastric lavage. The vomiting seemed to become less in amount for a time. This period of apparent improvement was followed by recurrence of symptoms in a more severe form. It was necessary to give daily infusions. An abscess developed, at the site of one of these infusions, in the left axilla. A second barium study did not give information of value concerning the upper gastro-intestinal tract but small flecks of barium given seven days previously were observed in the rectum, indicating that obstruction was not complete. The child passed small stools containing bile daily. He did not gain in weight but remained at about 6.6 lbs., being supported by daily infusions. No peristaltic waves were observed at any time and no tumor was made out by palpation. A diagnosis of partial obstruction in the duodenum either due to atresia or faulty attachment was suggested by the surgeons. Exploration was carried out June

19, 1934, under drop ether anesthesia. The stomach and the first portion of the duodenum were distended. The pylorus was normal. The right transverse colon was freed from its peritoneal attachments from the midline to the hepatic flexure. Further mobilization was made down to the cecum and the whole right colon was then retracted toward the left, allowing exposure of the root of the mesentery. There were a series of angulations in the retroperitoneal duodenum where peritoneal attachments produced an accordion pleated effect (Fig. 6). These bands were released and the duodenum traced until it emerged into jejunum under the mesenteric vessels. There were no signs of atresia. Closure was made in layers with silk.

Convalescence was complicated by continuation of vomiting in small amounts, but a gradual improvement was effected by careful regulation of his formula. He was discharged in good condition September 17, 1934, at which time he weighed 9.7 lbs. He was in the hospital in November, 1934, for otitis media. His weight at this time was 11.1 lbs. A bilateral myringotomy was done with relief. On February 28, 1935, he was admitted to the hospital because of a conjunctivitis and an acute purulent left otitis media. He was taking his food without trouble. His weight was recorded as 15.1 lbs. The baby was examined again on April 22, 1936. He was eating and sleeping well. He was regarded as normal. His weight was 22.6 lbs. at this time.

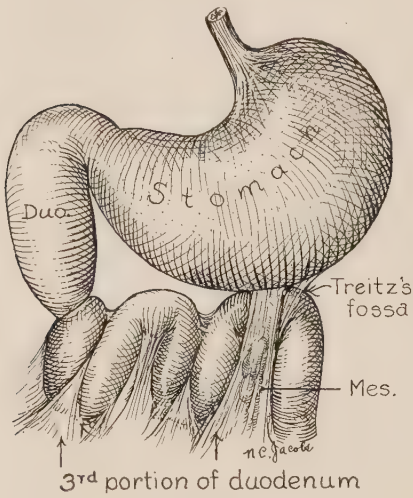

FIG. 6.—Condition found at operation in Case 9. This is an "accordion-pleated" obstruction due to hyperfixation.

Case 10.—No. 79531. M. M., a white female infant, third child, seven weeks old, prematurely born, was admitted to the Strong Memorial Hospital September 8, 1933, because of persistent vomiting since birth. Three hours after birth the baby "threw up red stuff" according to the mother. The character of the vomitus remained unchanged for four days, following which it consisted only of feedings previously taken. The symptoms persisted despite all possible changes in formulae and antispasmodics. The vomiting occurred immediately after each feeding and had consisted of a substantial portion of the feeding. Vomiting was definitely projectile. Constipation was marked, the infant having but one small yellow formed stool per day. The weight on admission was the same as the birth weight, 2,600 Gm.

Physical examination was entirely negative except for undernourishment and a palpable liver and spleen, each of which was 1 cm. below the costal margin. Laboratory examination showed some anemia; red blood cells, 3,700,000; hemoglobin, 80 per cent. The urine was negative. The Wassermann was negative.

All possible conservative measures were tried. The vomiting persisted, and even increased in frequency and amount. On one occasion four days after admission peristaltic waves were seen in the epigastrium passing from left to right. This was noted a second time on September 18. Gastro-intestinal fluoroscopy showed retention, no barium leaving the stomach during an hour and a half of observation. By this time the child's general condition appeared worse than on admission and laparotomy was performed for relief of pyloric obstruction.

Operation.—September 21, under ether anesthesia. A small right rectus incision was made. Because the stomach was dilated, the pyloric muscle was divided although no true tumor was present. Although an adequate explanation of the vomiting was not

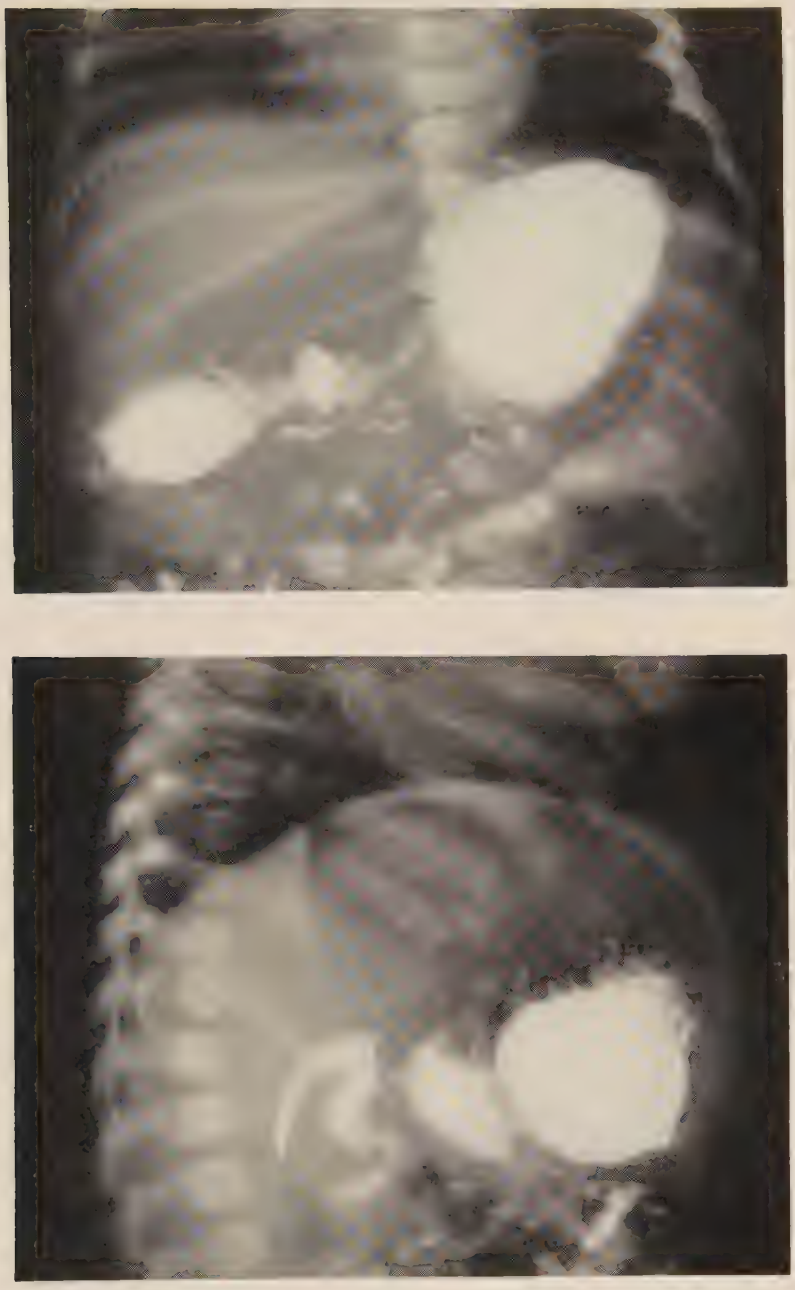

FIG. 7.—Case 11. Anteroposterior and lateral roentgenograms. Showing dilated stomach with folding of antrum and duodenal cap into posterior position. There is a twist of the normal duodenal curve.

demonstrated, the operation was terminated at this point because of the extremely poor condition of the patient.

Following operation the patient was given a transfusion and subcutaneous saline. During the first week after operation the patient's condition improved and vomiting was only occasional. However, beginning on the eighth day vomiting again became regular and frequent and on the following day coffee ground material, which gave a 3+ benzidine reaction, was vomited. A second transfusion was given. The patient vomited blood a second time. Her general condition was poor. The weight on October 3 was 2,550 Gm.

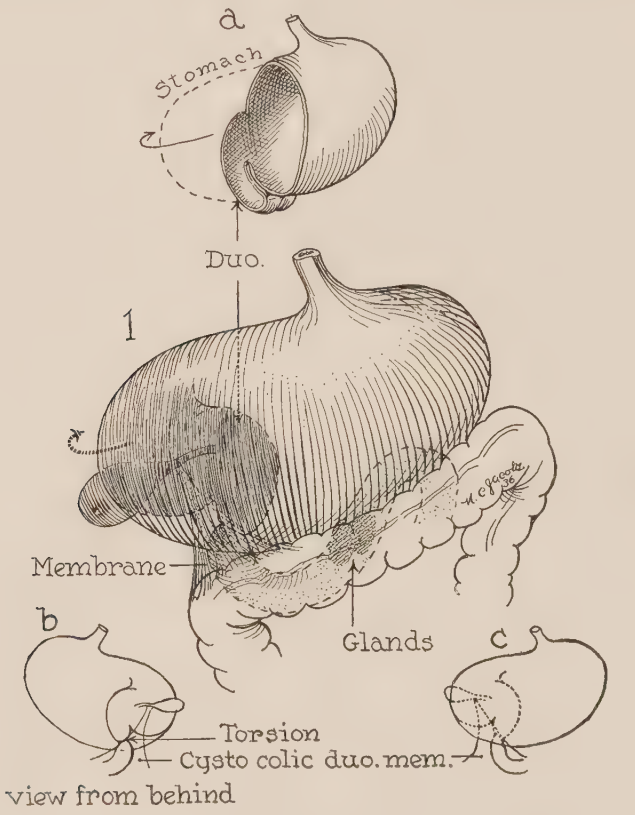

FIG. 7a.—Case 11. The condition found at operation explains the roentogenographic appearances. The obstruction in this case was due to a band running from the duodenum to the third part of the duodenum and colon.

A second laparotomy was performed October 4 under ether anesthesia, the previous incision being opened and considerably enlarged. The stomach was dilated. The cecum and appendix were in close approximation to the second portion of the duodenum to which they were attached by peritoneal bands which caused a kinking and obstruction of the duodenum. These bands were divided, releasing the cecum, which was placed in its normal position. A systematic exploration of the entire small bowel showed other peritoneal bands. About four inches from the ileocecal valve other peritoneal bands were found. These involved the ileum and transverse colon in a complicated knot which was released by division of the bands. About six inches proximal a similar condition was found. At Treitz's ligament there was found a triple twist of the bowel,

causing a loop to kink upon itself with an acute angulation when it entered a retroperitoneal position before emerging into the first portion of the jejunum. This portion of the intestine was intimately associated with the blood supply and consequently it was difficult to straighten it out. After correcting this last abnormality, it was noted that there was a three inch opening in the mesentery of the transverse colon, a congenital defect, as the edges were smooth and regular and contained the blood supply. This rent was carefully closed, following which the intestines were returned to the abdominal cavity and the wound closed.

The patient tolerated the operation extremely well considering her previous poor condition and the unavoidable shock associated with the manipulation. She responded well to an immediate transfusion. There continued to be vomiting but in general it was less frequent and regular and not projectile. The amount vomited was small. Her convalescence was retarded by numerous recurrent upper respiratory infections. In December bilateral otitis media was responsible for a temporary set back. Despite the occasional vomiting, however, the patient showed a general tendency to improve. Her weight increased slowly but steadily, on a moderately thickened formula, and at the time of discharge January 12, 1934, she weighed 4,300 Gm., an increase of 1,700 Gm. over her weight on admission. She was last seen a month after discharge, at which time she weighed 4,660 Gm. During the month she had vomited three or four times.

Case 11.—No. 110306. W. B., a baby girl, was admitted to the Strong Memorial Hospital December 26, 1935, because of vomiting. She was two weeks old at this time. The mother had had an uncomplicated pregnancy and no difficulties at delivery. The child's birth weight was 9 lbs. She was the tenth child in the family. Three of the children before this one had had vomiting, controlled by thickened feedings; one sister had died in infancy from vomiting and diarrhea of two months' duration after many different types of feeding had been tried without success. The present child was fed on the breast for only a few days. A breast pump was then used and the milk fed by bottle. On the tenth day a dilute whole milk formula was substituted for the mother's deficient milk supply. Vomiting was infrequent during the first two weeks. Every feeding was followed in 15 or 20 minutes by a moderate sized well formed stool. Five days before admission the baby became constipated. Stools were obtained only by glycerine suppositories. This child had lost one pound in weight. Examination showed nothing remarkable. She was given a modified formula and sent home. Within the next week she had diarrhea and a marked mastitis was present. She had projectile vomiting on January 7, 1936, and was readmitted January 8.

A suggestion of peristaltic waves in the epigastrium was made out by one observer and confirmed by several others during the next few days. She was placed on thickened feedings. Atropine was tried also but had to be discontinued. The baby continued to vomit and had diarrhea for several days. The symptoms then let up and she gained slightly in weight. She was discharged January 20, 1936. After being home for only a few days, there was a recurrence of symptoms. She regurgitated small amounts after each feeding; had six or more slimy movements a day; cried constantly when awake; and acted very hungry. The mother was reluctant to have her in the hospital but was worn out by the care of the baby during the next ten days. Accordingly the child was readmitted January 31, 1936. Peristaltic waves were easily made out after she had taken a few swallows of water. In a few days all her vomiting and diarrhea had been controlled. Arrangements were made to continue the treatment at home under supervision of a public health nurse. She was discharged February 6. She retained most of her food during the next ten days. And gained weight up to 10.2 lbs. But ten days later she had lost a little because of vomiting, with alternating constipation and diarrhea. A gastro-intestinal barium study was made March 2. She was three months old at this time and had not gained an ounce in the last month. The stomach was greatly distended and atonic. It folded back on itself over the pyloric region so that the antrum and pylorus were pointing toward the left and posteriorly. The duodenum occupied a peculiar twisted position. The

obstruction was partial since only half the barium remained in the stomach after four hours. The diagnosis of faulty fixation of the duodenum on the mesentery with partial obstruction to the stomach was made (Fig. 7).

At operation under drop ether anesthesia March 9 a greatly dilated redundant stomach was found. By gentle traction on the stomach the antrum and pylorus were exposed. The pyloric ring was in spasm which gave the impression of a hypertrophic stenosis, but it soon relaxed. The first portion of the duodenum was slightly larger than normal. On exposing the mesentery by freeing the transverse colon, it was evident that the large bowel had not migrated completely to the right side but was attached just beyond the midline in the region of the gallbladder. The gallbladder was large, and a well defined cystocolic membrane ran across it to the hepatic flexure region of the colon causing a torsion of the descending portion of the duodenum which was caught in this membrane. On freeing it the duodenum straightened out (Fig. 7*a*). The duodenum was traced all the way to its emergence from under Treitz's ligament without finding any other anomaly. Closure was made in layers with silk. Postoperative course was satisfactory. There was no vomiting. Feedings were resumed rapidly and the patient was discharged March 23, 1936. She weighed 11 lbs. at this time. She has had no symptoms since operation. She takes five to six ounces five times a day and is still not quite satisfied. Her weight at 4½ months, on April 16, 1936, was 12.4 lbs.

Symptoms and Signs.—The most constant symptom presented by every one of these patients is vomiting. It may have been noted a few hours after birth. Usually, however, it is observed only after the first feedings. The vomiting is usually fairly regular. It may occur immediately after a feeding or not till some time within three to four hours. In most cases it begins mildly but progresses to a more violent projectile type as time goes on. Vomiting may be irregular with free intervals of apparent improvement, or it may become steadily more frequent. The vomitus may consist simply in the ingested material, water, milk curds, mucus, or it may contain "coffee ground" material, or blood as noted in Cases 1, 2, and 11. In a doubtful case the presence or absence of bile in the vomitus has been used as evidence of an obstruction above or below the bile papilla. We have not found this examination very valuable; in fact, in this series, chemical or microscopic tests of the vomitus have scarcely ever been recorded.

The presence of blood or "coffee ground material" in the vomitus is considered pathognomonic of duodenal atresia or stenosis. On the other hand, the presence of bile in the vomitus is not a reliable diagnostic sign in localizing the obstruction as it has been demonstrated that enough bile can find its way through the microscopic openings to be readily detectable. Also, as Cordes pointed out, accessory bile ducts may be present entering the intestine above and below the obstruction. The detection of bile in the meconium is likewise subject to misinterpretation for the same reasons and also because of the fact that, while bile secretion begins normally about the third month of gestation, these congenital obstructions may not develop until much later. Consequently, despite a complete obstruction below the ampulla of Vater, the first bowel movements after birth may and often do contain bile which has reached the lower intestine before the establishment of the obstruction.

While normal meconium may be obtained, complete obstipation and absence of milk stools is the rule. If bile is present in appreciable amounts in the vomitus it aids in the differentiation, being absent in pyloric stenosis.

As a sequel to the vomiting there may result an upset in the bowel elimination. The usual trouble is constipation with a tendency towards small dry stools. Occasionally diarrhea has occurred; and alternating periods of constipation and diarrhea. In a number of cases the stool has been normal. Some of these obstructions are only partial in nature so that no absolute rule can be formulated. Blood was recorded in the stool examination in only one instance.

When the vomiting has been persistent, the baby either loses weight rapidly, or he fails to gain under the best of supportive treatment. The weight loss in some instances has been as high as one-quarter of the original birth weight in ten days to two weeks. But it is remarkable that these newborn babies can be carried along for a considerable period, just as adults can be in high obstructions, provided that adequate water and salt are supplied. In some cases where the vomiting has not occurred after every feeding the babies have even shown some slight gain in weight. This is usually a testimonial to the care and skill of the attending pediatrician.

Upon examination the signs of dehydration are frequently observed. These babies appear puny and cyanotic when severely ill. The infants may also show other developmental abnormalities (Cases 1, 6, and 7), or may be prematurely born (Cases 5 and 10). Peristaltic waves passing from left to right have been seen at some stage in examination of these infants in a majority of instances. In Case 1 a tumor was visible occupying a transverse position. When vomiting has been severe there may be signs of alkalosis, manifested by tetany and convulsions.

Course.—The pediatricians attempt to regulate the feedings of these babies by varying their formulae; substituting different materials; changing the frequency or consistency of the feedings; feeding by tube; expressing air from the gastro-intestinal tract and refeeding; and the use of antispasmodics. In some instances the babies are found to be habitual air swallowers and regurgitators and must be broken of the habit. Rarely, there also seems to be a protein sensitiveness to milk in some infants. Fluids and salts can be supplied parenterally as necessary when the vomiting is not controllable by other means. Transfusions may be of value. We cannot praise too highly the skill and resourcefulness of our pediatric staff in the management of these infants. When, in spite of all efforts, vomiting continues, an attempt to localize the lesion accurately by roentgenologic examination seems warranted.

Roentgenologic Examination.—Although it is admitted that a barium mixture should not be used above an obstruction, we have made use of a thin mixture of barium in most of these cases. The information which it gives us in this group seems to outweigh its disadvantages as we have decided to operate usually in any case because of the persistence of symptoms. The

barium can be evacuated by lavage or by vomiting when operation has to be delayed for any reason. In Cases 1, 2, and 4 characteristic dilated atonic stomach and duodenum shadows indicated that the obstruction was near the third portion of the duodenum (Figs. 1, 2, and 4). In Case 3 a peculiar sausage shaped shadow along the lesser curvature attracted our attention. This child was thought to have hypertrophic pyloric stenosis until the roentgenogram helped us visualize the true lesion. The parallel shadows of this herniated loop can be seen through the gastric shadow on this one film of the series (Fig. 3). Roentgenograms in Cases 5, 7, and 9 showed delay in gastric emptying but gave no other help in the diagnosis. In Case 8 the dilated duodenum showed retention and a clear rounded outline after the barium had left the stomach. It clearly indicated that the partial obstruction was in the duodenum behind the stomach (Fig. 5). In Case 11, by use of the lateral view, it was possible to recognize that the greatly dilated stomach had folded back upon the pyloric antrum; and that there was a peculiar angulation in the duodenal loop leading away from this region (Fig. 7). We think that this information together with the fact that it sometimes shows that obstruction is not complete is of value to us. We have never yet seen any deleterious effects from the use of this method.

Differential Diagnosis.—The only real problem in diagnosis is to differentiate these cases from those of congenital hypertrophic pyloric stenoses. Congenital hypertrophic pyloric stenosis usually occurs in males and usually in the first born child in the family. The onset of vomiting is rarely at birth. It usually appears some time during the second to the sixth week. In a majority of cases the vomiting increases in severity, becoming projectile in character. Peristaltic waves passing from left to right can be made out in about three-quarters of the cases. A palpable tumor can be felt with varying success, this depending to some extent upon the importance attached to it by the examiner. The other symptoms and signs—loss of weight, dehydration, alkalosis, constipation and scanty urine are common to the two conditions. A study of congenital hypertrophic pyloric stenosis in our clinic showed that it occurred in males in 90 per cent; that it had been noted in the second child in the family once; in the third child twice; and once in the sixth child of the family; that vomiting started at birth in one instance; and that it did not start till two months of age in another; the rest of the cases following the rule; that peristaltic waves were observed in 80 per cent; and that a palpable pyloric tumor was made out in just half of the cases. In our cases of congenital obstruction there were seven females to four males. The majority also were not the first born in the family. There was one first born girl and three other possible first born babies though this point was not stated in the history. The others ranged from the third to the tenth child in the family. The vomiting came on early—usually before the first week, sometimes during the first day, and often following the first ingestion of anything. Two of the children were prematurely born; and three showed other congenital abnormalities. Peristaltic waves were made out in

seven of the 11 cases but no tumor was palpated in any of them. The roentgenogram had diagnostic value in six of the 11 cases. It would appear from this review that we should look with suspicion upon any baby which starts vomiting during the first week of life, especially if of the female sex and with other signs of congenital anomalies. If no relief can be obtained by medical measures we should be prepared to give a thin barium mixture for roentgenologic appraisal of the cause. The presence of peristaltic waves with the absence of a palpable tumor might be regarded as of some significance in helping to make the diagnosis. The presence of bile or blood in the vomitus would be in favor of some lesion other than hypertrophic pyloric stenosis.

Intussusception can be ruled out by its usual onset still later in life than that of hypertrophic stenosis; and by the typical acute picture it presents.

Strangulated inguinal hernia must sometimes be differentiated but in our experience this has not been difficult.

Prognosis.—Intestinal obstruction in the new born due to congenital abnormalities is not uncommon. The fact that a large percentage of such cases reported in medical literature is based on postmortem examination is evidence of the extremely high mortality associated with this condition. A universal appreciation of the comparative frequency of its occurrence will tend to improve results as the unfavorable outcome in many instances is directly attributable to incorrect diagnoses or to diagnoses established too late for surgical intervention to be successful.

In a certain percentage of cases of intestinal atresia (15 per cent according to Davis and Poynter), the congenital defects are multiple and beyond the aid of surgery. Furthermore, many of the defects occur in the duodenum and upper jejunum (194 of the 392 cases in the series of Davis and Poynter), thus placing them in the category of high intestinal obstructions. This, superimposed upon the fact that the new-born infant is not a good operative risk, mitigates against improvement in results in such cases. Admitting a necessarily high mortality upon the basis of these facts, there still remains a definite percentage of fatal results which can, conceivably, be eliminated by early correct diagnosis and the prompt institution of surgical treatment. An organic mechanical obstruction demands early operation for its relief. Since many of these congenital defects are amenable to operative treatment and this fact can be established only at the time of operation, all cases so diagnosed should be given the opportunity of relief which only operation affords.

The prognosis in cases of congenital occlusion of the small intestine is extremely poor. The first successfully treated case is credited to Ernst[14] who operated upon an infant ten days old for congenital duodenal atresia, relieving the condition by a "duodeno-entero-anterior anastomosis." Eleven months postoperatively the patient was progressing normally. In 349 published articles there are reports of 29 cured cases: 15, in which the obstruction was due to malrotation, and 14, in which the obstruction was intrinsic,

atresia or stenosis.[5] In the 60 cases reported by Ladd the mortality was 70 per cent. The mortality in cases of malrotation was 55 per cent (nine cured of a total of 20 cases) and in cases of intrinsic obstruction it was 80 per cent (eight cured of a total of 40 cases). These results showed a great improvement when one considers the statement of Thorndike[13] who placed the mortality of congenital duodenal atresia at 99 per cent. In the present group of 11 cases there has been only one death, a mortality of 9 per cent.

The prognosis in cases of congenital anomalies about the mesentery depends to a large extent upon the alertness of the attending physician. These babies can stand a considerable amount of fluid and salt loss if it is compensated for by parenteral medication. It is also true that in some cases the obstructions are only partial so that the baby gets some nourishment. When the child loses weight or fails to gain because of the amounts lost by vomiting, steps should be taken to correct the underlying fault if possible. The surgeon also must bear his share in getting these babies by the operation. This requires gentle handling of tissues more than any other single thing. The fate of these babies depends then upon the preoperative condition—a reflection of the care given by the attending physician; the operative manipulation and judgment—a reflection on the care of the attending surgeon; and the continued combined efforts of both physician and surgeon in the postoperative follow up. In our series the results have been excellent. All the babies have been carried through the operation to relieve their obstructions without a fatality. There have been two deaths, one almost two years after the operation, from pneumonia; the other as the result of a second operation for cleft palate two weeks after the duodenal obstruction had been relieved satisfactorily. This baby was a great feeding problem which forced action against better judgment. The postmortem examination showed bronchopneumonia which may well have been due to aspirated food.

Treatment.—Preliminary preparation for operation is exceedingly important. Loss of weight and dehydration must be combated by restoring the water balance. This may be done by the intravenous, intraperitoneal or subcutaneous routes. Blood, water, glucose and salt may be used as circumstances require. We usually fluoroscope these infants to see whether an enlarged thymus is present. If so, operation would be delayed for a few days following roentgen therapy. Usually no preoperative drugs are necessary. Occasionally atropine in 1/400 to 1/200 gr. doses is given. We have used local infiltration in the past but when there has been crying, straining and evisceration of the bowels, so much damage has been done in restoring the viscera to the peritoneal cavity that we have abandoned this practice. Drop ether carefully given by a well trained anesthetist, we believe to be the anesthetic of choice. A very small quantity will often suffice. Most writers agree that babies do not stand hemorrhage, shock or infection well.[19, 20, 21] For these reasons during operation there must be most careful hemostasis; gentleness in handling tissues; lack of exposure to prevent heat loss; and progression without delay so that a minimum of anesthesia is necessary.

Emphasis should not be placed on speed in these operations. Babies will stand operations well for one and one-half hours or longer if necessary. However, the more time that a surgeon can save, without sacrificing gentleness, the better. Respect for the tissues is by far the most important factor in these operations. The flashy, speedy, rough surgeon will not do well with these patients. Abdominal closure is made in layers with fine silk. We have had no disruption of these wounds and no infection. The dressing is made light and sealed in with waterproof adhesive.

In congenital absence of a segment of duodenum, obviously the relief of the obstruction by operation is the only treatment possible. A short circuiting operation, duodenojejunostomy or gastro-enterostomy, is the logical procedure. The choice between these two procedures will vary with the operator and the conditions encountered in the individual case. In those cases in which there is a diaphragm stretched across the lumen of the bowel,

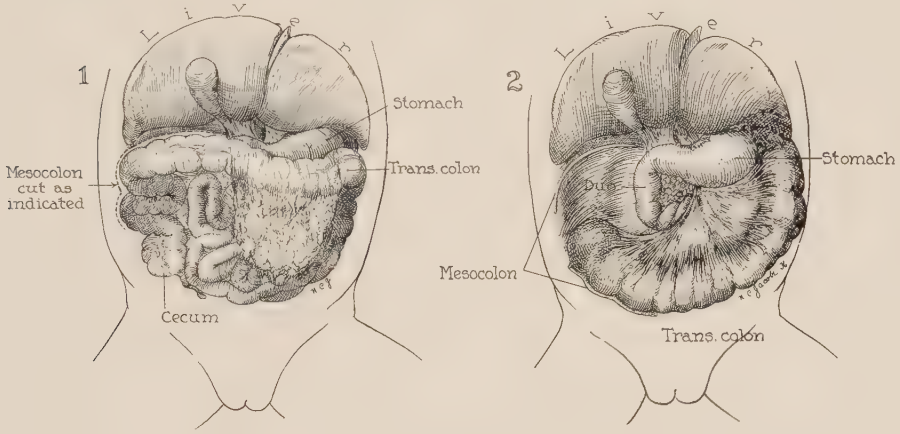

FIG. 8 (1 and 2).—Represents the operative approach to the root of the mesentery. The transverse and ascending colons are freed from their lateral attatchments and the retroperitoneal duodenum exposed. The condition illustrated in 2 was present to some degree in Cases 5, 6, 7, and 10.

longitudinal incision of the intestine, removing the diaphragm and transverse suturing, as proposed by Morton[15] and performed by Petersen,[16] should be seriously considered as being a simpler procedure than intestinal anastomosis. However, it is often difficult to definitely establish the presence of such a diaphragm. Under no circumstances should an enterostomy (jejunostomy) be performed, as it is only a palliative procedure which is not well tolerated by infants. In those cases in which it has been done a rapid death resulted. Clogg[17] has made a valuable suggestion that, in as much as congenital intestinal defects are prone to be multiple, one should inject fluid into the collapsed bowel below the obstruction, forcing it along to discover the presence of any additional defects. This possibility should always be kept in mind, as it might easily condemn to failure what otherwise would be an entirely successful operation.

The operation of choice for the extrinsic anomalies due to faults in migration, descent, and fixation, is not to attempt a gastro-enterostomy or

entero-enterostomy as has been done so often in the past. The surgeon should realize that this is the type of obstruction which can be successfully untangled if he knows how to get at it. The best way to unravel these puzzling anomalies is to get a clear view of the mesentery. This can be done only by detaching the transverse and ascending colons from the parietal wall and rotating them toward the midline, which gives excellent access to the root of the mesentery (Fig. 8). This operation we had worked out independently in our clinic only to find that Ladd had been using the identical procedure. We believe that this operation will result in a decided improvement in the operative mortality in these extrinsic anomalies. It stands in much the same relationship as the division of the pyloric sphincter did to cases of pyloric stenosis with the consequent abandonment of the previous posterior gastro-enterostomy operations.

The surgeon should not enter the abdominal cavity with a preconceived idea that he is going to undertake a certain procedure. It is most difficult to abandon the plan to fit circumstances which may not demand it. It is especially difficult not to wish to simply divide the pyloric muscle in some of these cases.

After operation special nursing care is of great importance. The baby must be kept warm, but not so hot that he will lose his fluids through perspiration. Cold draughts must be avoided. Fluids should be restored again. Codein may be used for pain. Distention should be combated with stupes, rectal tubes and later enemata. Babies do not stand pain and distention well. Their reactions to these are violent and alarming. Lavage may also be necessary. Acidosis may be prevented by the use of glucose. The baby should be restored to his normal environment as rapidly as his condition warrants. His dressings must be kept dry, especially during the first week. Measures should be taken to seal off the urinary tract from the operative site by suitable apparatus. It is a pleasure to acknowledge the close cooperation of Dr. Samuel Clausen and his staff in the after care of these patients.

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SPLENECTOMY FOR HEMOCLASTIC CRISES

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DURING the clinical course of certain blood diseases, namely, congenital hemolytic icterus and thrombopenic purpura, there occur episodes of increased blood destruction. These are known clinically as "crises." They may develop spontaneously or be precipitated by various environmental factors. In congenital hemolytic icterus the increased destruction affects chiefly the erythrocytes. In thrombopenic purpura there may be an increased destruction of thrombocytes. Although splenectomy is ordinarily the treatment of choice in the more chronic stages of these hemoclastic dyscrasias, it has been thought,[7, 9, 11, 12, 13] and it is the accepted surgical teaching[4, 5, 6, 8, 10] at the present time, that the acute exacerbations are a direct contra-indication to splenectomy.

Previous studies within our group[1, 13, 14] have provided evidence which incriminates the spleen as one of the principal factors in the physiologic control as well as in the pathologic destruction of the circulating blood elements. If such be the case, then, during exacerbations of splenic activity, even to the extent of "crisis" proportions, removal of the spleen is not only desirable, but in many instances may become essential to survival. Nevertheless, the risk involved in undertaking any major abdominal operation with a total red count rapidly approaching the one million mark, has, heretofore, interdicted splenectomy under such circumstances.

In addition to the conviction that the major rôle was played by the spleen in the diseases under discussion, our early studies revealed, as a part of the postsplenectomy mechanism, an immediate marked postoperative rise in both the red blood cells and platelets, which amounted essentially to an autotransfusion. The specificity and significance of this response have been discussed[1] and form the basis for our having undertaken a series of splenectomies during active hemoclastic crises without as yet encountering one operative fatality.

It is the purpose of this paper to present the pertinent clinical evidence from eight patients each successfully splenectomized during an acute hemoclastic crisis. Six of the patients had congenital hemolytic icterus; two had thrombopenic purpura.

CASE REPORTS

Congenital Hemolytic Icterus

Case 1.—H. S., a mail carrier, age 50, was admitted to the Research Service of the University Hospital, December 4, 1933, presenting splenomegaly, severe epigastric pain and deep jaundice.

As a boy he had noted his sallow skin. He was advised also of an anemia, and was aware that he fatigued easily. About 20 years ago he was diagnosed as having "splenic anemia." He had already noted the mass in his upper left quadrant. He was hospitalized, about 19 years ago, for what appears to have been a subacute exacerbation. Splenectomy was advised at that time but was not accepted. Since then, he has presented the characteristic picture of chronic congenital hemolytic icterus. During the past three years the disease has gradually become more severe, and in addition, mild symptoms of cholelithiasis have appeared.

On November 29, 1933, while delivering mail, he experienced acute, severe epigastric pain, which disabled him. Jaundice became evident the next day, and grew progressively deeper. His food intake was decreased and an unusual weight loss ensued. An associated acute hemoclastic crisis was suspected, and he was referred to the Research Service by Dr. Paul A. Adams.

His past history reveals mainly the story of congenital hemolytic jaundice with the characteristic ebb and flow of its minor symptomatology. During the past few years his appetite has diminished and an associated weight loss has ensued. To his knowledge none of his family except his son has had evident icterus. This son, an icteric, frail boy of 15, with marked splenomegaly, was subsequently (July 12, 1934) successfully splenectomized on the Research Service for this same disease.

Physical examination of the patient revealed marked weakness, enlarged spleen, anemia and a deep jaundice. T., 98.2°; P., 66; R., 15; B. P., 140/70. The sclerae were deep yellow. The spleen extended 4 cm. below the umbilicus. The liver was enlarged and tender. There was definite right epigastric tenderness. The urine was negative, other than for the high bile content. The stool was not acholic and was negative for blood. Roentgenologic examination revealed a small annular shadow, about 1 cm. in diameter to the right of the upper body of the third lumbar vertebra. The gallbladder was not visualized.

The significant blood findings are presented in Chart 1. There was no significant anemia (red blood cells, 3,820,000; the reticulocytes were 4 to 6 per cent); a microcytosis (mean red blood cell diameter 6.9, mean red blood cell volume 100 cu. microns), and an increased fragility of the red blood cells (0.582 to 0.300) were found. The icterus index was 882, the highest we have ever recorded. The patient was Type B blood group. On December 5, the direct van den Bergh was immediately positive, and the indirect was 21 mg. During the 48 hours subsequent to admission the red count fell to 3,000,000 and the reticulocytes gradually increased to 14.2 per cent. The icterus index decreased appreciably, to 428 (Chart 1).

The findings thus indicated an icterus of double origin; an older, congenital hemolytic form with splenomegaly, and a more recent acute, acquired form due to obstruction by a common duct stone. Since about two-thirds of patients with hemolytic icterus develop pigment calculi within the gallbladder, such an occurrence is readily understandable and should be anticipated. There was evidence of only moderate hemolysis. The preoperative icterus index was 440, predominantly of obstructive origin.

Two courses of management were considered: (1) Splenectomy, to stop an impending hemoclastic crisis, and (2) a preliminary cholecystostomy, in an attempt to relieve the acute and severe cholemia. The latter seemed the more threatening. It was clearly realized, however, that the cholecystostomy might provoke even more severe hemolytic activity by the greatly enlarged spleen.

The relatively good level of the red cells and hemoglobin (Chart 1), together with

the evidence of but moderate hemolysis, influenced us to follow the second course in the hope that the obstructive jaundice might subside somewhat before the onset of a more acute hemolysis. The patient was prepared for cholecystostomy by the administration of 5 cc. of 10 per cent calcium chloride in 200 cc. of normal saline intravenously, and by a hypodermoclysis of 1,500 cc. of Ringer's solution containing 5 per cent glucose. This was given the evening before, and repeated the morning of the laparotomy.

Cholecystostomy was accomplished uneventfully on December 7, under local anesthesia, using a nitrous oxide-oxygen supplement. The abdomen contained considerable bile-stained ascitic fluid. The liver was greatly enlarged. It was greenish, finely nodular and the lobules were unusually prominent. The gallbladder was enlarged, and its walls were thickened and injected. It compressed readily, and contained numerous stones. The cystic duct was about 7 Mm. in diameter when flattened. It appeared to be patent. The common duct was tense and distended. At its lower portion, well above the ampulla, was a hard, irregular mass about 15 x 10 x 10 Mm. This was thought to be a stone, although it was not explored with a needle. The spleen was huge and moderately adherent. The splenic capsule was thick, white and opaque. The adhesions were soft and vascular.

Opening the gallbladder fundus revealed the thick, fibrous wall, a rather normal yellow-brown bile and numerous characteristic pigment stones, which varied up to 18 Mm. in diameter. They were black, somewhat elastic, friable and most irregular in shape. They were removed and the cystic duct explored. It was patent. The gallbladder was drained. Careful hemostasis was effected, and there was but a moderate loss of blood. The patient was returned to his room in good condition with the blood pressure 78/44.

Progressive relief from the cholemia ensued. The icterus index, 24 hours postoperative, fell to 120 (Chart 1). On the other hand, hemolysis definitely increased. On the evening of December 9, 36 hours postoperative, the red count had fallen to 1,610,000 and the hemoglobin to 3.9 Gm. The reticulocytes had risen to 20 per cent. An acute, even fatal, hemoclastic crisis threatened.

From our previous experience it seemed evident that splenectomy was indicated as being the most reasonable procedure to arrest the unusually severe, although expected complication. We could also, with reasonable assurance, depend upon the customary postoperative autotransfusion as a restorative measure. An emergency splenectomy was performed, therefore, December 9. The patient was prepared for this as for the cholecystostomy. Local anesthesia, supplemented by nitrous oxide-oxygen, was employed. Adrenalin, 1 cc., was administered intramuscularly about 20 minutes before clamping the splenic pedicle. This was to ensure maximum contraction of the smooth muscle in the splenic capsule and trabeculae.

The spleen was removed without incident through a left subcostal Kocher incision. During the procedure the blood pressure, increased by adrenalin, remained elevated around 140/50. There was no evidence of circulatory failure and prompt postanesthetic recovery ensued.

The spleen weighed 1,740 Gm., and measured 22 x 15 x 9 cm. It was firm, and apparently contracted. The capsule was in places as thick as 4 Mm. and an opaque white. The splenic pulp was increased. It was dark red, cellular, friable and more fibrous than normal. Microscopically the splenic corpuscles were infrequent, and the pulp spaces were filled with blood. The phagocytic clasmatocytes were increased.

Immediately following operation the characteristic and expected autotransfusion ensued, and the reticulocytes decreased (Chart 1). The icterus index continued to decrease, and reached 30 on December 14. Convalescence was then uneventful until December 13, when the patient became disoriented, arose and walked about the room. A chill followed, and later a fever and leukocytosis (Chart 1). Vomiting supervened. There was no bile in the vomitus. *B. coli* infection of the splenectomy wound was apparent December 15. It was drained and treated by dakinization. The skin flaps separated and on December 22 a secondary closure was undertaken under local anesthesia.

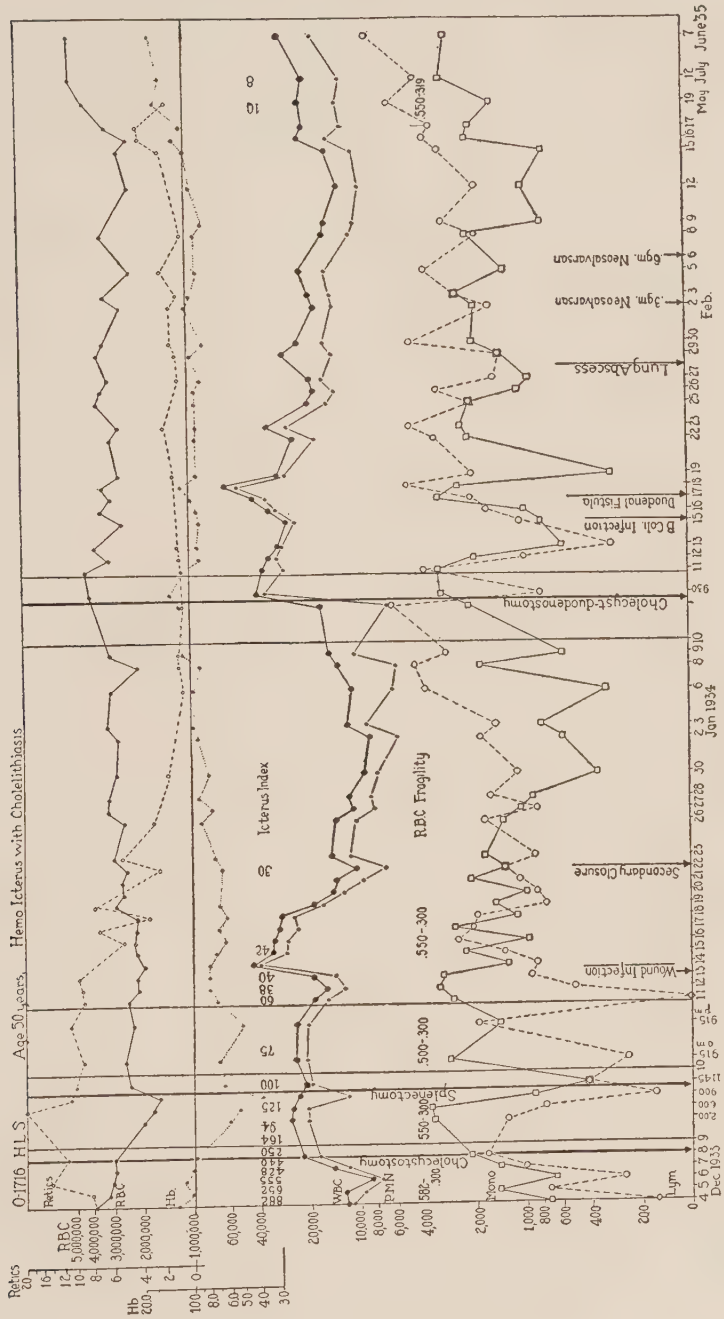

CHART 1.—(Case 1) Splenectomy for the hemoclastic (erythroclastic) crisis of congenital hemolytic jaundice. (Journal A.M.A.)

Dakinization was continued and sound healing, without subsequent herniation, eventually occurred.

Biliary drainage continued; however, there was no evidence of autodigestion about the cholecystostomy opening. The original rubber tube was later replaced by a mushroom catheter which minimized the leakage. Convalescence was steady and afebrile.

On December 27 the biliary tract was visualized by injecting lipiodol through a small catheter inserted into the cholecystostomy opening. The gallbladder, also the cystic, hepatic and common ducts were examined fluoroscopically. The lipiodol came to an abrupt stop, 6 cm. down the dilated common duct. Roentgenograms revealed an irregular block in the region of the shadow which was inferred to represent the common duct stone. There was a questionable leak past the stone into the intestine. A subcutaneous tract was also demonstrated connecting the cholecystostomy margin with the infected splenectomy wound. This was possibly the original avenue of infection. No reaction followed this investigation.

The bile draining from the cholecystostomy showed no evidences of containing pancreatic secretion, which apparently entered the duodenum normally as shown by aspiration through a duodenal tube. Thus an irregular, occluding common duct stone remained, definitely above the ampulla of Vater and which did not obstruct the external pancreatic secretion. The increased risk of removing this stone, by mobilizing the duodenum medially, performing a choledochotomy, and of possibly inserting a T tube, was weighed. To be sure, the cholecystostomy assured temporary biliary drainage. Eventually, however, owing to the continued fluid and mineral loss, it required closure. Cholecystoduodenostomy was finally elected as the best solution of the problem. This was accomplished January 10 under local anesthesia, using supplementary nitrous oxide-oxygen.

The cholecystostomy opening was dissected free, the gallbladder fundus mobilized, and the stoma trimmed. The gallbladder fundus was then attached to the adjacent duodenum and a two row cholecystoduodenostomy accomplished. The suture line was then fastened to the fascia of the gallbladder bed with interrupted silk sutures. A Penrose drain was left just under the skin. Ephedrin was given at the end of the operation. The blood pressure was 88/66. The pulse was fair, rate 112. There were no evidences of shock.

During the ensuing two days the temperature rose to 104.8°; pulse, to 160; and increased respirations. These all gradually subsided. On January 14 the patient expectorated a foul smelling, thick, tenacious mucus. On January 15 a *B. coli* infection of the right abdominal wound was noted. This broke down and on January 17 a duodenal fistula became established. Bile and duodenal juice were identified. The skin edges became eroded. The duodenal fistula was treated by plugging, strapping, care of the skin edges and the use of a light cradle. It soon began to fill in and was closed by February 6.

On January 19, cough and considerable foul smelling sputum production were noted. This continued. A roentgenologic examination January 26 revealed a left hilus shadow suspicious of lung abscess. This was confirmed by a stereoscopic examination January 31. A fluid level was evident. The abscess wall was thin. Adequate postural drainage was instituted. Because of the character of the sputum on February 2 and again on February 6, neoarsphenamine was administered. The sputum production began to decrease February 4. It was only 10 cc. in amount by February 10, and ceased on February 12. Stereoscopic examination, February 16, showed no evidences of any residual lung abscess, and a clear parenchyma. General improvement ensued. The patient's appetite returned, and he gained weight. The patient was discharged February 17.

At no time during this stormy convalescence, subsequent to the splenectomy, was the hemolytopoietic equilibrium seriously disturbed. The bone marrow met all demands promptly and efficiently as they arose (Chart 1). There were no evidences of decompensation or incapacity. This is of significance in evaluating the fundamental nature of congenital hemolytic icterus.

Since leaving the hospital the patient has gained 40 pounds (18 Kg.). Both

operative wounds are well healed without herniation. There have been thus far no evident gastro-intestinal symptoms referable to the results of the extensive abdominal surgery. The patient resumed his occupation as mail carrier, June 1, 1934. July 12, 1934, the day splenectomy was successfully accomplished on his son, the father's red blood cells were 4,850,000, hemoglobin 14.2 Gm., color index 0.95, reticulocytes 1.2 per cent, icterus index 8, hematocrit 52, erythrocyte fragility 0.550-0.319 and leukocytes 19,000, with an essentially normal differential percentage. After two and one-half years the patient continues to remain clinically well.

Case 2.—B. Y., a housewife, aged 56, was admitted to the University Hospital February 9, 1935, presenting great weakness, an unusual pallor, moderate jaundice and an enlarged spleen.

Since childhood she had noted that she very easily became fatigued, that even minor ailments disabled her greatly, and that she had a peculiar pallor. At one time she was thought to have pernicious anemia. Four months ago she developed an upper respiratory infection. This resulted in an increase in the anemia and the development of recognizable jaundice. During the succeeding interval there have been remissions and relapses with, however, a progressive increase in weakness and pallor. One son of 28 has had manifest jaundice during the past year. There is evidence of generalized arteriosclerosis. A cerebral vascular accident two years ago resulted in a residual hemifacial paralysis with ptosis of the eyelid and some atrophy of the right side of the tongue.

Physical examination revealed a striking exhaustion. She was poorly oriented. The loose, pale skin indicated considerable weight loss. Pitting edema was noted over the tibiae and sacrum. The retinal arteries were tortuous and striped. Hearing was diminished, particularly on the right. The bony thorax was somewhat immobile with a decreased vital capacity and other evidence of pulmonary emphysema. Crepitant râles were constantly heard over the posterior right base. The heart revealed an occasional premature ventricular systole. The pulmonary second sound was accentuated. A short, soft blowing systolic murmur was audible over the base. T., 98.0°; P., 100; R., 15; B. P., 110/60. The palpable arteries were moderately sclerosed. The enlarged and firm spleen extended to just below the umbilicus.

Neurologic examination by Dr. Harry LeFever revealed a right facial weakness (supranuclear); weakness of the left masseter and pterygoid muscles; paresis of the right upper eyelid; deviation of the tongue to the right; impaired hearing on the right with bone conduction greater than air conduction; and unequal reflexes. These findings were interpreted as the result of a previous brain stem lesion, probably a hemorrhage.

Laboratory investigation confirmed the characteristic findings of congenital hemolytic icterus; an anemia, red blood cells 1,480,000, hemoglobin 5.0 Gm.; a reticulocytosis of 54 per cent; microcytosis; and increased fragility of the red blood cells, .319–471. The icterus index was 60. There was a leukopenia, 3,300 to 3,800. The blood N.P.N., glucose, and carbon dioxide combining capacity were normal. The urine revealed no bile but pathologic amounts of urobilin; it was otherwise normal. The stools contained bile pigment but occult blood was not demonstrable. The Wassermann and Kahn tests were negative. The direct van den Bergh test was negative; the indirect was 4.2 mg.

Upon this evidence a diagnosis of congenital hemolytic icterus, without biliary obstruction, seemed warranted. On the other hand, destruction of the red blood cells was gradually becoming more marked. During the first four hospital days the erythrocyte count decreased from 1,620,000 to 1,310,000, while the icterus index rose from 60 to 75; and the reticulocyte count from 54 to 70 per cent. These findings signified increasing hemolysis and indicated the onset of a "crisis," and an unfavorable course of the disease. The blood typing showed Group O; however, from our previous studies transfusion seemed contra-indicated. Since at this time three emergency splenectomies for hemoclastic crises had already been successfully accomplished, we had less hesitancy in advising this operative procedure as a means of arresting the threatening hemolytic process.

Kidney function tests were within normal limits. An E.K.G. revealed a normal

sinus rhythm with an occasional sino-auricular premature contraction; and left ventricular preponderance. Roentgenologic examination of the chest revealed increased bronchovesicular markings. A flat film of the abdomen revealed evidence of gallstones.

Splenectomy was accomplished uneventfully February 13. Preoperatively morphine gr. ¼ and atropine gr. 1/150 were administered. The usual left rectus incision was made after local novocaine infiltration anesthesia; and nitrous oxide-oxygen was administered during the exploration and splenectomy. Considerable yellow tinged ascitic fluid was encountered. The distended, thick-walled, opaque, and moderately adherent gallbladder contained three large calculi. Palpation of the biliary ducts revealed no evidence of stones. A small accessory spleen 15 Mm. in diameter was removed from the posterior splenic pedicle.

The spleen weighed 811 Gm. and measured 23 × 16 × 6.5 cm. Supravital studies of the pulp revealed the usual increased phagocytosis of the red blood cells by clasmatocytes, atrophy of the lymphatic elements, and engorgement of the splenic parenchyma by erythrocytes. Grossly and microscopically the pathologic appearance was that of congenital hemolytic icterus. The usual postoperative autotransfusion was immediately apparent. Within the first 24 hours the erythrocytes had increased to 2,030,000 and the hemoglobin to 5.8 Gm.; with 11,900 leukocytes. Other than for some productive cough, subsequent convalescence was steady and uneventful. The icterus diminished and gradually disappeared. The reticulocyte count decreased. The patient's strength slowly returned and she was discharged March 6, 1935, 21 days postoperative, greatly improved.

Case 3.—O. L., a white housewife, age 52, was admitted to the Research Service June 7, 1935, presenting a purulent otitis media complicated by a progressively increasing anemia and a recently developed icterus.

A little over a year previously, while under hospital observation for goiter, a marked leukopenia and a moderate, sustained anemia and thrombopenia had been discovered. The white blood cells ranged from 1,200 to 2,500; the erythrocytes from two to three million. During the past year various medical regimens had been instituted in an attempt to overcome this status, with little apparent result. It was believed that her bone marrow was moderately hypoplastic. About two weeks ago, however, she developed an acute purulent otitis media, which ruptured the right tympanum, with a resultant profuse foul smelling discharge. Two days before admission a right facial palsy supervened. With the establishment of this infection more evident anemia developed, accompanied by definite clinical icterus for the first time. The leukopenia became more pronounced.

Upon arrival at the hospital the total leukocyte count was only 200. Hemolytic streptococci were cultured from the middle ear discharge. Direct smears showed contaminating bacilli and cocci. The icterus index was 40. The direct van den Bergh was negative; the indirect showed a trace. The red blood cell count was 1,200,000, with 18 per cent reticulocytes. The platelets were greatly diminished.

Owing to the atypical picture which she had presented during the past year, suggesting a hypoplastic bone marrow, a transfusion of 400 cc. of citrated blood was given, cautiously, soon after admission. This was followed by a chill and a severe reaction.

Upon physical examination a splenomegaly, hitherto absent, was discovered. The fragility test revealed some decrease in the resistance of the erythrocytes to hypotonic solutions. The icterus index had increased to 60. With evidence now available of an increasing hemolytic anemia, of increased erythrocyte fragility, of a rising reticulocyte count, of splenomegaly, of a mounting icterus index and of an even more marked leukopenia—the conclusion that we were dealing with an atypical congenital hemolytic icterus seemed warranted. Further investigation strengthened the view that we were now facing a definite hemoclastic crisis initiated by the middle ear infection, and splenectomy was determined upon.

On June 8, 1935, a 920 Gm. spleen was removed without incident under local anesthesia with supplementary nitrous oxide-oxygen. The liver and gallbladder appeared to

be normal, as did the other viscera. No accessory spleens were seen. Upon supravital study the splenic pulp was found to be greatly engorged with neutrophilic leukocytes, in sharp contradistinction to the extreme peripheral leukopenia. The phagocytic clasmatocytes were greatly increased in number.

Convalescence was complicated by the development of a large rectal abscess with surrounding necrosis from which hemolytic streptococci, similar to those recovered from the otitis media, were cultured. This was treated by hot moist packs, by incision and drainage; and the danger of general sepsis was combated by repeated daily small blood transfusions (from June 11 to June 16). The rectal area healed slowly.

On July 3, 1935, the erythrocyte count was 3,610,000 with only 1.4 per cent reticulocytes. The leukocyte count, however, remained low—1,800—for some time. The patient was discharged July 6, 1935, definitely improved. She has been followed during the past year. On July 8, 1936, just one year after leaving the hospital, her blood picture showed: red blood cells, 4,250,000; hemoglobin, 13.0 Gm.; reticulocytes, 0.2 per cent; leukocytes, 5,100. This restoration to normal of the previously depressed blood findings is further evidence that we were dealing with the splenomegaly of congenital hemolytic icterus.

Case 4.—L. H., a white housewife, age 31, was referred to the Research Service by Dr. C. C. Fitzpatrick of Jackson, Ohio. She was admitted January 1, 1936, critically ill; and presented the picture of an acute hemoclastic crisis, *in extremis.*

Her early history is that of unrecognized congenital hemolytic icterus. She was in apparent good health up to about four years ago, when she had a cesarean section to terminate her third pregnancy. At that time an anemia and a splenomegaly were noted. Liver therapy was tried without appreciable effect. She was twice hospitalized and transfused. About a month ago liver therapy was resumed and six injections of liver extract were given. This resulted in some questionable improvement. One week ago, however, she was confined to bed because of increasing anorexia and weakness. Two days ago persistent nausea and vomiting developed. The resultant extreme dehydration and demineralization complicated the clinical picture on admission to University Hospital, with evidence of recent rapid loss of weight, of increasing anemia, with moderate jaundice and progressive dyspnea.

Physical examination revealed a critically ill patient. The skin was pale and icteric; the mucosae and finger tips were cyanotic; there was moderate dyspnea with orthopnea; no petechiae or other evidences of bleeding could be found, yet the anemia was profound. The sclerae were yellow. T., 99.6°; P., 122; R., 32; B.P., 118/50. The pulse was full. A blowing systolic murmur was audible over the entire precordium. The liver was moderately enlarged. The spleen extended to the iliac crest, and was tender. A pitting edema was demonstrable over the tibial crests. There were no abnormal neurologic findings.

Immediate examination of the blood revealed red blood cells, 800,000; hemoglobin, 2.1 Gm.; reticulocytes, 16 per cent; leukocytes, 10,800; platelets greatly decreased, and an icterus index of 60. The urine showed an albuminuria. The N.P.N. was 40 mg. per cent; the blood glucose 160 mg. per cent; the plasma CO_2 30 vol. per cent; and the blood chlorides 205.

Intranasal oxygen was instituted. This resulted in some improvement of the cyanosis and dyspnea. A small indirect transfusion was given about three hours after admission. This was accompanied by chilliness, temperature elevation and some reaction. Later sodium bicarbonate and glucose were given intravenously and after four hours 500 cc. of citrated blood. Only slight transitory improvement was noted.

The relative lack of response in this critically ill patient, during the first 12 hours of medical treatment, left no alternative but splenectomy. While the rationale of this operative procedure had been established in our previous experiences, no patient had presented heretofore quite so critical a condition. It was decided to explore and limit the procedure to ligature of the splenic artery, in case adhesions or operative difficulties made the actual removal of the spleen too hazardous.

On January 2, 1936, without any preoperative medication, 150 cc. of novocaine containing eight drops of adrenalin per 100 cc. was infiltrated along the left upper rectus region. The spleen, exposed through an adequate left rectus incision, was large, bluish and fortunately nonadherent. Nitrous oxide-oxygen was then administered. The spleen was luxated and its pedicle freed. One-half cubic centimeter of adrenalin was next injected directly into the splenic artery. A definite, visible contraction of the spleen followed, thus squeezing blood cells from the large splenic reservoir into the circulation and effecting an autotransfusion. The pedicle was then clamped and tied and the spleen removed. Several accessory spleens, ranging from 3 Mm. to 3 cm. in diameter, were then removed from the region of the pedicle.

Immediate improvement occurred in the patient's clinical status, which allowed the abdomen to be thoroughly explored. The liver was enlarged. Its lobules were evident; however, it did not appear to be cirrhotic. The gallbladder was enlarged, firm, thickened and contained numerous irregular stones, which are so characteristic of the calculi occurring in instances of congenital hemolytic icterus. The ducts appeared to be patent. The stomach and colon were normal, and the pelvis was negative. The patient left the operating room definitely improved.

The spleen weighed 1,635 Gm. and measured 26 x 15 x 10 cm. Grossly it was characteristic of hemolytic icterus. Microscopic sections revealed a diffuse fibrosis and numerous dilated endothelial channels. Increased phagocytic activity was evident, as well as increased blood pigment. The splenic corpuscles were atrophic.

Postoperatively the blood pressure remained over 100 systolic. Hematology: red blood cells, 1,710,000; white blood cells, 23,700; hemoglobin, 3.9 Gm.; platelets, 350,000. Hypodermoclysis and intranasal oxygen were administered. At the commencement of the second day 600 cc. of citrated blood were given to hasten the clinical recovery. From that time on convalescence was progressive and uneventful.

On February 22, three weeks postoperative, the blood findings paralleled the clinical improvement. Red blood cells, 4,490,000; hemoglobin, 13.4 Gm.; white blood cells, 7,200; reticulocytes, 0.4 per cent; and platelets 1,890,000. The icterus index was also normal. The patient was discharged January 28. Six months later, on July 22, she was clinically well. Red blood cells, 5,420,000; hemoglobin, 14.9 Gm.; reticulocytes, 4 per cent; leukocytes, 8,400; icterus index, 6.2; and thrombocytes, 1,900,000.

Case 5.—G. P., female, age 4, with an acute severe hemoclastic crisis, was splenectomized September 6, 1933, by Dr. V. A. Dodd.[1]

Case 6.—V. T., female, age 20, with an acute hemoclastic crisis, was splenectomized March 25, 1935, by Dr. Luke Zartman.[1]

Thrombopenic Purpura

Case 7.—E. M., a colored schoolgirl, age 17, was admitted to the University Hospital June 23, 1934, for the management of purpura with an attendant severe anemia. Unusual pallor together with scattered subcutaneous and submucous hemorrhages were evident.

She had been well and active up to early May, when unusual bleeding from the gums was first noted. This became more marked. Menstruation early in June was prolonged and profuse. An increasing pallor, with the symptoms of progressive anemia, caused her to consult a physician. She was hospitalized elsewhere June 14, and the presence of a severe anemia confirmed. A blood transfusion was given June 16. This resulted in but temporary relief, since the metrorrhagia continued and in addition epistaxis and hematemesis developed.

Her past history revealed a long recognized tendency to easy bruising. Nevertheless, she had undergone tonsillectomy eight years previously without any complications. No other incidence of unusual bleeding was known to have occurred in her family.

Upon entering the hospital the patient was dyspneic, somewhat cyanotic and perspiring profusely. T., 103°; P., 120; R., 48; and B. P. 103/?. Her pallor was striking.

Cutaneous petechial hemorrhages up to 5 Mm. in diameter were evident. The eyes reacted normally and the sclerae were clear. There were no retinal hemorrhages. The gums were spongy and hemorrhagic. Ulceration had developed about a carious and abscessed lower right molar, and had extended into the adjacent tongue and soft palate. Necrosis with resultant fetor oris was evident. Recent bleeding was also noted in the nasal cavities. The heart was negative, other than for a blowing systolic murmur heard at the apex. Pelvic examination revealed a cervical polyp and uterine bleeding. Other examinations were negative. The spleen was not palpable, and apparently not enlarged.

Investigation of the blood (Chart 2), revealed 1,770,000 red blood cells, hemoglobin 4.9 Gm. (Newcomer), color index 0.9, reticulocytes 57 per cent; erythroblasts 1,200; and normoblasts 2,400 per cmm.; mean erythrocyte diameter 8.6 microns with both anisocytosis and poikilocytosis extreme on direct examination but with no "sickling," immediate or delayed; erythrocyte fragility was markedly decreased, hemolysis started at 0.319 and was not complete until something less than 0.210 salt equivalent; icterus index 6; leukocytes 30,000, with 67 per cent actively motile, mature, neutrophilic granulocytes, 5 per cent myelocytes C and metamyelocytes, 1 per cent eosinophils, 18 per cent lymphocytes, and 9 per cent monocytes; blood platelets 3,500; bleeding time 23 minutes; coagulation time (Howell) 11 minutes; calcium time 12 minutes; prothrombin time 12 minutes; no clot retraction in 24 hours; tourniquet test (50 Mm. of mercury) showed innumerable petechiae after ten minutes. The Wassermann and Kahn tests were negative; carbon dioxide combining power 36; blood Group O. Doubtless due to the oral infection and anemia the daily temperature ranged from 99° to 104° F. Blood cultures were negative.

The striking thrombopenia together with the coagulation findings pointed to the diagnosis of idiopathic thrombopenic purpura hemorrhagica. Elsewhere a diagnosis of aplastic anemia with symptomatic purpura had been entertained. However, both the red and white cells in the circulating blood revealed such evident regenerative responses that an aplastic bone marrow was at once ruled out. The unusually high reticulocyte count (57 per cent) even resembled that seen occasionally in congenital hemolytic icterus, or in pernicious anemia after liver therapy. A sternal bone marrow puncture yielded no added information. The nucleated red cell response (3,600 per cmm.) added further evidence of unusually active erythropoiesis. Even after an epinephrine test June 26 (Chart 2), blood platelets did not appear in the peripheral circulation. In fact, platelets were essentially absent throughout the entire period of preoperative observation (Chart 2).

Intensive medical treatment was first instituted. During the first 24 hours 50 cc. of whole blood were given intramuscularly. On the second day 500 cc. were given intravenously. Viosterol, a high fat-protein diet, large amounts of orange juice daily, oral hygiene, and even cauterization and packing of the cervical canal were of little avail, since the hemorrhagic tendency and anemia steadily increased. Splenectomy was finally advised, but was refused until the fourth hospital day when the red blood cells following a profuse hemorrhage fell to 725,000 and the hemoglobin to 2.4 Gm. The nucleated red cells had increased to 6,000. The leukocytes were 25,000, the reticulocytes 30 per cent and *no platelets were to be found in the peripheral blood.* An emergency transfusion of 700 cc. of citrated blood was given to the comatose and moribund patient, which increased the red blood count to 1,230,000. A second of 400 cc. was given later in the day. The following day (June 28) the general clinical condition of the patient was greatly improved. Red blood cells, 1,660,000; hemoglobin, 4.9 Gm.; leukocytes, 10,800; platelets, 0; T., 99.6°; P., 88; R., 24; and B. P., 100/58. It was thereupon decided to resort to surgery without further delay and to attempt ligation of the splenic artery, if splenectomy seemed inadvisable.

On June 28 splenectomy was accomplished uneventfully with a minimal loss of blood. A left rectus incision was made under local anesthesia, which was supplemented by nitrous oxide-oxygen, for the intra-abdominal manipulation. No free fluid was

encountered in the abdomen. Petechial hemorrhages, however, were numerous and were increased by the necessary handling of the viscera. The spleen was normal in size, appearance and position. Its pedicle was short. It weighed 225 Gm. At the conclusion of the operation the pulse was 96, respirations were 24, the blood pressure was 140/90; red blood cells 1,990,000, reticulocytes 15 per cent, hemoglobin 5.9 Gm., leukocytes 14,800, and blood platelets 21,900. Twenty-four hours after the splenectomy the leukocyte count was 52,000 with 85 per cent active mature neutrophils, red blood cells 2,230,000, hemoglobin 6.2 Gm., and blood platelets 89,000 (Chart 2). All bleeding had ceased.

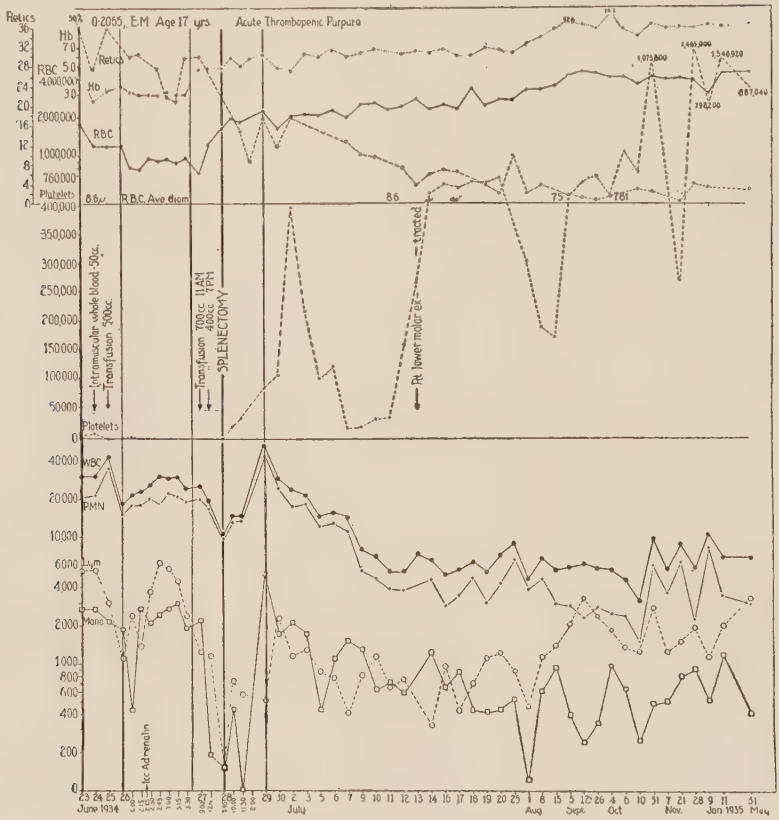

CHART 2.—(Case 7) Splenectomy for the hemoclastic (thrombocytoclastic) crisis of thrombopenic purpura. (Journal A.M.A.[1])

Investigation of the fresh splenic pulp by the supravital technic failed to reveal free extracellular clumps of blood platelets. The débris in the clasmatocytes presumably represented whatever of these elements had reached the spleen. This permitted an explanation for the failure of platelet increase subsequent to the preoperative injection of epinephrine. Microscopically sections of the spleen revealed a suggestion of chronic inflammation. The splenic corpuscles were prominent but normal. The clasmatocytes did not appear to be increased in number or unusually phagocytic. The reticulum was hypoplastic; the endothelial cells and the vascular bed were not remarkable. The splenic pulp was not engorged with red blood cells as occurs in hemolytic icterus, nor with blood platelets, as is described in certain purpuric states.

Immediately following the splenectomy the blood platelets increased in number until on the fourth day postoperative they attained a count of 395,000. During this period

they varied greatly in individual size. During the succeeding eight days a gradual decrease ensued without notable change in the patient's condition. By the twenty-second postoperative day they had reached 748,000 (Chart 2).

With the cessation of the hemorrhages and the attainment of satisfactory convalescence the ulceration of the mouth cleared rapidly. The abscessed molar was extracted on the fifteenth postoperative day and the gums were then given needed attention. Both the hemoglobin and red cells gradually increased. At the end of three weeks there were 3,500,000 erythrocytes. The reticulocytes promptly decreased (Chart 2). The bleeding time became normal, clot retraction occurred promptly and the tourniquet test (ten minutes at 50 Mm. mercury pressure) failed to cause petechiae. By the ninth day postoperative the elevated temperature and pulse had returned to normal. The patient was discharged July 20, on the twenty-second postoperative day, in good condition.

During the past two years she has maintained an entirely normal existence, with a platelet count sustained consistently at about one million per cmm. (Chart 2). She

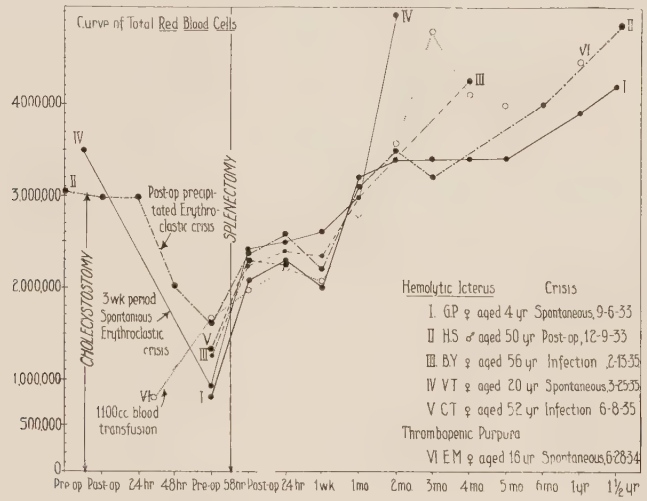
Curve of Total Red Blood Cells
4,000,000
3,000,000
2,000,000
1,000,000
500,000
0
CHOLECYSTOSTOMY
SPLENECTOMY
Post-op precipitated Erythroclastic crisis
3 wk period Spontaneous Erythroclastic crisis
1100cc blood transfusion
Hemolytic Icterus Crisis
I. G.P. ♀ aged 4 yr. Spontaneous, 9-6-33
II. H.S. ♂ aged 50 yr. Post-op, 12-9-33
III. B.Y. ♀ aged 56 yr. Infection 2-15-35
IV. V.T. ♀ aged 20 yr. Spontaneous, 3-25-35
V. C.T. ♀ aged 52 yr. Infection 6-8-35
Thrombopenic Purpura
VI. E.M. ♀ aged 16 yr. Spontaneous, 6-28-34
Pre-op Post-op 24 hr 48 hr Pre-op 5 8 hr Post-op 24 hr 1 wk 1 mo 2 mo 3 mo 4 mo 5 mo 6 mo 1 yr 1½ yr

CHART 3.—The results of splenectomy for hemoclastic crises in six cases. (Journal A.M.A.[1])

has married and, on March 21, 1936, gave birth to a normal female child. Throughout this stay under observation in the University Hospital, from March 20 to 30, 1936, no evidence of recurrence of the purpuric state was noted. The platelet count remained high. There was no unusual bleeding either previous to or after labor. The puerperium was quite normal. She continues to remain clinically well.

Case 8.—A. J., a housewife, age 61, with an acute hemoclastic crisis occurring in the course of thrombopenic purpura, was successfully splenectomized June 30, 1936. She was dismissed in good condition on July 30. This splenectomy was accomplished after the completion of this paper.

CONCLUSIONS

The evidence presented by these eight patients, all successfully surviving a major surgical procedure during a hemoclastic crisis, further supports the rationale of splenectomy in the treatment of properly selected and prepared patients suffering from hemolytic icterus or thrombopenic purpura of splenic origin. There have, thus far, been no failures. We attribute this success to careful differential diagnostic studies, optimum preoperative management

and the application of modern surgical knowledge to the problem. The implications of this study add materially to our knowledge of the pathologic physiology of the spleen.

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THE INFLUENCE OF HEREDITY IN CANCER

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FROM earliest times there must have been thought and speculation in regard to the effects of heredity on human life. Biblical history states that all Israel was reckoned by genealogies.[1] Heredity plays an important rôle in the size of individuals, their physical shape or habitus, muscular strength, color of the hair and eyes and in many other characteristics. It is only a short step farther to the realization that a definite tendency to disease is also transmitted in certain families. Hemophilia, diabetes and cardiovascular disease illustrate this and there are many others.[2, 3]

The question of an hereditary tendency in cancer patients has evoked much discussion. According to Kurbler,[4] Paget (1857) found such a taint in 22 per cent of 254 cancer patients, but Winiwarter (1878), in 548 cancer patients in Billroth's clinic, found it in only 1 per cent. Siegrist gives 2.5 per cent and Baker (1866) as high as 43 per cent. Most of these observations were from the figures of insurance companies and obviously the conclusions are not to be considered reliable. The differences are too great. As recently as 1908 Bashford[5] made a statistical analysis of the records of the Imperial Cancer Research Fund and concluded that cancer is probably always acquired. Until our records for many generations are more perfect than at present, the statistical approach to this problem will be suggestive only.

In an analysis of family histories at the Eugenics Record office of the Carnegie Institution of Washington, Little[6] shows that the progeny of a cancerous mother and noncancerous father were found to contain an excess of cancer. His study "seemed to show beyond any question that heredity plays a rôle in determining cancer incidence in man."

Wells[7] states that it seems safe to maintain that the existence of an hereditary influence on the susceptibility and resistance to cancer has been established for both man and animals. Maud Slye[8] also believes this to be the fact and suggests that cancer susceptibility behaves like a recessive, insusceptibility like a dominant characteristic. Macklin,[9] using a different statistical approach, very logically shows that the family in which a mother and three daughters had their breasts removed for cancers before the age of 21 years had a 100 per cent incidence of the cancer, while the figures for 1928 in Canada for cancer of the breast at that age show the normal incidence would be 1 in 1,090,000. She feels that there are numerous families in which the incidence of cancer is so high that it precludes any explanation that it is by chance alone that these persons were affected. History and medical literature are full of strikingly similar instances.

Cancer is said to have been frequent in the family of Napoleon. Napoleon himself, his father, his brother and two sisters were all victims of cancer. Warthin's family S shows 38 members dying of cancer through four generations. Finney[10] reports a family in which the mother, four daughters and three nieces have all been operated upon for cancer. All but one had cancer of the breast. Three of the daughters had a second cancer in the other breast, and one of these had a third cancer in the stomach. There are, however, 37 other members of the present adult generation of sisters, brothers and cousins who are free from cancer. The proportion of diseased individuals and the location of the cancer seem to remove the element of chance. Kurbler[4] describes a cancer family. In the second generation, one woman and two men died of cancer of the stomach. One woman also died of uterine and one woman of hepatic cancer. A son of the woman who had gastric cancer died of cancer of the rectum and all three daughters of the cancer of the liver case had cancer of the breast. In the fourth generation, there were also two women with cancer of the breast. The author recently removed the breast of a woman 38 years old. Her sister had had a radical mastectomy at the age of 36, followed a year later by removal of the other breast, and died of cancer six months later, at the age of 38. Pack[11] cites two brothers age 52 and 54 who were both in the hospital at the same time with adenocarcinoma of the stomach. One came in for tumor and pain, while the other showed loss of weight only. A roentgenologic study demonstrated the growth in both cases. Very convincing evidence is submitted by McFarland and Meade,[12] who feel that the simultaneous occurrence of similar, symmetrical tumors in both of homologous twins tends to prove the genetic origin of such tumors. They have collected a series of 40 tumors occurring symmetrically and simultaneously in 20 pairs of monozygotic twins. These were equally divided between benign and malignant growths. These authors[13] describe carcinomata of the stomach occurring simultaneously in uniovular twins, who were 70 years old. Their symptoms were identical, and the tumors were symmetrically located in the stomach. There was rapid progression of the disease and a similar pathologic report. Both tumors were adenocarcinomata of the cardia and fundus which extended nearly to the pylorus. The symptoms started in October, 1933, in one, and in November, 1933, in the other. One died on April 29, 1934, and the other on June 17, 1934. Croon[14] described adenocarcinomata of the uterus in twins who were both 50 years of age.

Deelman,[15] after an investigation of the family history of 350 cases of cancer and 250 control cases, concludes:

(1) "In a series of families of cancer patients, cancer is more frequent than in any other series of families.

(2) "The surplus of cancer in the family of cancer patients always accumulates in certain distinct families.

(3) "There is a large group of cancer patients in whose family cancer

absolutely is not more frequent than agrees with the 'normal' chance of dying from cancer.

(4) "Hereditary influences are only evident in a small category of cancer cases."

Hunter,[16] using more modern statistics from insurance company records, draws the same conclusions as Deelman but states them differently. He says, "There are undoubtedly families which show a strong predisposition to the disease (cancer), but these are rare in proportion to the total population. So far there has been no conclusive proof that a person with one death in a family, either a parent or brother or sister, need fear a predisposition to the disease." It has been generally accepted that mice transmit all the Mendelian factors necessary for susceptibility to transplanted tumors and also that hereditary factors determine the characteristics of both the host and the tumor cell.[17] Macklin[18] has summarized the subject very well and concludes in part that:

(1) "Cancer is due to an inherited factor.

(2) "Cancer of a specific type, in a specific organ, at a specific age, tends to occur in families.

(3) "Related persons were affected ten times as often as unrelated persons.

(4) "Chronic irritation, when present, appears to accentuate the speed of a reaction which was destined to occur at a later date.

(5) "Identical twins, who have the same type of tumor, in the same organ and frequently at the same age, afford excellent proof of the rôle which inheritance plays in the production of cancer.

(6) "Belief in the inheritance of cancer affords an opportunity for early diagnosis."

She analyzed the life history of a family, later described, in which the mother, three daughters and a son all died of carcinoma of the rectum. Using the 1931 vital statistics for Canada, she found "123 deaths from rectal cancer between the ages of 30 and 55, the ages in which your group died. There were 104,517 deaths that year from all causes, at all ages. Therefore, approximately one in every 750 persons was dying from rectal cancer at those ages. Now, if cancer were due to chance, the chances for rectal cancer affecting five persons in the same family between the ages of 30 and 55 would be determined by the binomial theorem, as follows: (a plus 750b).[5] If that is expanded, you find that in 237,304,687,500,000 families of five persons, you will get one family where all died of rectal cancer without there being any necessity for heredity being brought in as the explanation. If heredity is the explanation, and if it were due to a dominant factor, then, with a mother showing it and a family of four children, you would find the chances for all four affected varying from one in one, if the mother were homozygous for rectal cancer, to one in 16, if she were heterozygous. The chances would be also one in 16 for all children affected, if cancer were due to a recessive, for in this family, the mother would show it and the father would be hetero-

zygous for it, and the results would be the same as if it were due to a dominant with the mother having one factor for it, in other words, heterozygous." Maud Slye was recently quoted as having stated that, in her opinion, cancer could be conquered only by breeding it out of the race. She estimated it would take 500 years to accomplish this.

Abridged Histories of a Family Personally Observed

Case Reports.—The mother, Sophie R., in 1916, at the age of 54 complained of frequent, small, lumpy stools and a sensation of pressure in the rectum and back. Examination showed a small cauliflower growth at the lower end of the rectum. A colostomy was performed but death ensued on June 11, 1916. A microscopic examination was not obtained.

A daughter, Sophie R., age 24 began in 1913 to lose weight and complain of frequent bowel movements accompanied by a griping pain in the region of the transverse colon. She also noted blood in the stool. In June, 1914, an appendicostomy was performed with plication of an incompetent ileocecal valve. She gained weight and the diarrhea was controlled, but traces of blood were still present. A tuberculin test was positive. In September, 1914, a rectovaginal fistula developed and a large nodular mass was felt in the lower rectum. A microscopic examination showed a malignant adenoma. Radium was used but death ensued on March 30, 1916, at the age of 30 years.

A daughter, Bertha R., age 25, noted a mass the size of a grapefruit in her abdomen. On June 3, 1915, the terminal ileum, cecum and a retroperitoneal fibroma were removed. In June, 1931, she reported that for several months she had noticed a slight change in her bowel action. First there was a diarrhea and now for two weeks there have been gas and distension. Her age is now 40. Examination showed a hard distended abdomen and a circular cauliflower mass, admitting only the tip of the index finger, was felt three inches up the rectum. A colostomy was performed. Microscopic examination of the growth showed an adenocarcinoma. Death occurred on the day following operation.

A son, William R., age 31, in October, 1930, stated that six months previously he had suffered from several attacks of indigestion accompanied by chest pain and gas in the stomach. One month later looseness of the bowels ensued and pain in the left loin with much intestinal gas. Examination showed a large nodular growth of the rectum forming a complete circle. There were also metastases in the lung. On August, 9, 1931, a colostomy was performed. Microscopic examination showed an adenocarcinoma. He died April 1, 1932.

A daughter, Emma R., age 47, the sole surviving member of the family, was admitted to the Presbyterian Hospital, New York, on December 10, 1935. She had had a mucous colitis for many years. Three months before admission her bowel movements, which had previously been two or three a day, changed to six or eight a day. Only once had she noticed a little blood in the stool. Examination showed a mass 10×5 cm. about the umbilicus. There was also a large fungating mass 4 cm. inside the anal sphincter, involving the anterior and left side of the rectal wall, with partial obstruction. A colostomy was performed. Microscopic examination showed carcinoma of the rectum, with metastases to the liver and mesentery. She died on December 28, 1935.

Environment has been considered a factor in the production of cancer. Various members of this family lived at seven different known addresses.

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ACUTE PERFORATION OF GASTROJEJUNAL ULCER *

REPORT OF TEN NEW CASES AND A REVIEW OF NINETY-THREE COLLECTED CASES

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THE term gastrojejunal ulcer is used in this paper to include all secondary ulcers situated at or adjacent to anastomoses between the stomach and jejunum irrespective of their gastric, marginal or jejunal location. The qualifying term acute perforation sometimes is used loosely in the literature to include subacute perforation, gastrojejunocolic fistula or fistula through the abdominal wall. It is restricted here to the use ordinarily made of it in this country with reference to perforation of peptic ulcer which corresponds to the German expression acute perforation into the free peritoneal cavity. Whereas ulcers which occur in the region of gastrojejunal anastomoses seem to possess the etiologic and pathologic features of primary peptic ulcer, they are to be distinguished from the latter by virtue of their occurrence in a small but definite proportion of cases as a sequel to surgical anastomosis. Synonyms include anastomotic ulcer, marginal ulcer, secondary jejunal ulcer, ulcus pepticum jejuni and peptic jejunal ulcer. Gastrojejunal ulcer is to be differentiated also from the rarely occurring primary jejunal ulcer.

Eighteen years after Woelfler[99] (1881) performed the first successful gastrojejunostomy, Braun[6] (1899) reported the first case of gastrojejunal ulcer. A point of interest here is that the first case of gastrojejunal ulcer to be reported was one in which acute perforation took place. Goepel[25] reported the first successful suture of acute perforation of gastrojejunal ulcer in 1902. In one of the earliest comprehensive papers on the subject, Paterson,[65] in 1909, originated the term gastrojejunal ulcer. At that time, ten years after the first case was reported, he stated that there were 52 authenticated cases of gastrojejunal ulcer on record.

ETIOLOGY.—*Incidence.*—Because it is known that many of the observed cases have not been reported, it is readily admitted by those who have written on this topic, that it is not possible to determine the frequency of occurrence of gastrojejunal ulcer. There are numerous casual references in the literature to cases of acute perforation of gastrojejunal ulcer. It is doubly difficult, therefore, if not impossible, to determine the incidence of this complication of gastrojejunal ulcer. Published estimates of the occurrence of gastrojejunal ulcer vary from 1.4 (Walton[95]) to 34 (Lewisohn[50]) per cent. In 129 cases of gastrojejunal ulcer collected by Lieblein,[53] there were 30 cases of acute perforation, or an incidence of 23 per cent. If we accept Sokolov's[82] and Lahey and Jordan's[48] estimates that 1,000 cases of

gastrojejunal ulcer have been reported in the world literature, our search of the literature, which yielded 93 more or less complete case reports, would indicate an incidence of acute perforation in gastrojejunal ulcer of less than 1 per cent. Singer and Meyer[81] point out that there is a relatively large group of unrecognized cases of free perforation which, if added to the known cases, would increase the percentage of incidence materially.

Contributory Etiologic Factors.—Until the problem of the etiology of primary peptic ulcer is solved, it is not likely that the cause of secondary gastrojejunal ulcer will be found. Some of the factors contributory to the production of gastrojejunal ulcer, which seem to have a bearing on acute perforation, will be briefly reviewed.

Despite our lack of knowledge regarding the direct or ultimate cause of gastrojejunal ulcer, two very important facts stand out. One is the peculiar susceptibility of the jejunum (the tissue susceptibility factor of Ochsner and his co-workers[63]) to the influence of gastric juice. The other is the marked tendency of gastrojejunal ulcer to penetrate or perforate. The latter is attested to by the high frequency of subacute perforation of gastrojejunal ulcer and by the frequent occurrence of gastrojejunocolic fistula.

Many of the factors which seem to contribute to the incidence of uncomplicated gastrojejunal ulcer also appear to contribute to the frequency of complicating acute perforation of gastrojejunal ulcer. Factors on the part of the patient are constitutional predisposition (Ochsner, *et al.*[63]), early age, male sex, high concentration of free hydrochloric acid, coexisting infection, and failure to follow postoperative medical management. Technical considerations which should be avoided by the surgeon are anterior gastrojejunostomy, artificial occlusion of the pylorus (von Eiselsberg pyloric exclusion), entero-anastomosis (Braun's or Roux's en Y), the use of the Murphy button and non-absorbable sutures. Gastrojejunal ulcer and in turn acute perforation has been reported but it is rare following resection of the stomach for malignant disease. It appears, therefore, that the same factors which contribute to the production of benign peptic ulcer seem to be operative in the formation of secondary ulcer.

The most common site of gastrojejunal ulcer is in the efferent limb of the jejunum.

Pathology.—The pathogenesis of acute perforation of gastrojejunal ulcer is apparently the same as that of acute perforation of primary peptic ulcer except that the jejunal mucosa is more susceptible than that of the stomach or duodenum to the proteolytic effects of gastric juice.

The essential gross and microscopic pathology is the same as that of primary peptic ulcer. The ulcer which perforates acutely usually is single but may be multiple. Except in gastrojejunocolic fistula, the tendency to heal is less common than in gastric or duodenal ulcer. There is a marked tendency toward recurrence manifest in some perforating gastrojejunal ulcers. An outstanding feature, and the one which chiefly concerns us here, is this tendency to penetrate and involve other tissues. Based on this characteristic,

gastrojejunal ulcers may be divided into three groups: (1) Non-penetrating ulcer which, while it may erode a blood vessel or obstruct the stoma of the anastomosis, does not extend beyond the wall of the stomach or jejunum. (2) Chronic penetrating ulcer, which may form an indurated mass involving the transverse mesocolon, the colon, the omentum or the anterior abdominal wall with or without abscess formation, or which may penetrate into the colon or through the abdominal wall with fistula formation. (3) Acute perforating ulcer, which allows the escape of gastric or jejunal content into the free peritoneal cavity. This tendency to acute perforation of ulcer is so marked in some patients that in one group acute perforation of gastric or duodenal ulcer is found to have preceded the performance of gastrojejunostomy whereas in a second group multiple successive perforations of gastrojejunal ulcer have taken place.

Symptoms and Diagnosis.—Acute perforation of gastrojejunal ulcer may or may not be preceded by suggestive prodromal symptoms. When premonitory symptoms are present, they usually consist of recurring dyspepsia appearing from a few days to as long as 25 years after operation. The dyspepsia often is refractory to the usual alkali and dietary management. In gastrojejunal ulcer the pain is usually on the left of the midline and at or below the level of the umbilicus.

In acute perforation of gastrojejunal ulcer as in peptic ulcer the pain is usually sudden and severe. The subjective symptoms are so characteristic that in several recorded instances patients who have experienced previous perforations have made their own diagnosis. On examination, the greatest tenderness and rigidity may be in the left lower quadrant of the abdomen because the fluid which escapes through the perforation passes downward on the left of the vertebral column. The late symptoms and signs are those of perforative peritonitis.

In those patients in whom the evidence points to acute perforative peritonitis, the diagnosis of acute perforation of gastrojejunal ulcer is made chiefly on the history of the patient's having had a gastrojejunostomy performed. The most difficult but least important condition to be differentiated is perforation of recurrent gastric or duodenal ulcer, the primary and confirmatory signs of which, as recently shown by one of us (H.L.T.[91]), are characteristic in definite proportions of cases. In these latter conditions the recurrent symptoms more nearly simulate the original ulcer distress.

Prognosis.—The prognosis in acute perforation of gastrojejunal ulcer, treated or untreated, is grave. Most of the untreated cases are fatal. Wright[102] reported a mortality of 23 per cent in cases treated by simple suture with 10.4 per cent of good results and 31 per cent of which required further operation.

Treatment.—The prophylactic treatment of acute perforation of gastrojejunal ulcer begins with the treatment of gastric or duodenal ulcer, the principles of which are well known. Most students of gastrojejunal ulcer

are agreed that gastrojejunostomy should be restricted to cases in which pyloric obstruction is present and in which the acid values are low.

Except in late cases wherein progressive peritonitis has supervened, the active treatment of acute perforation of gastrojejunal ulcer is surgical. In late diffuse peritonitis the injudicious application of surgery may be not only harmful but fatal. With respect to treatment in early cases, it may be said that there are two schools of thought. The adherents to one school maintain that simple suture is safest and therefore sufficient for the primary operation and can be followed by medical management and by radical surgery later if required. The members of the second school believe that in selected cases curative measures are warranted. If the duodenal ulcer is healed and the pylorus is patent, the gastrojejunostomy may be taken down and normal continuity restored. If active peptic ulcer is present, pyloroplasty, gastroduodenostomy to the first or second portion of the duodenum or Pólya or Billroth partial gastrectomy may be employed.

It is hardly necessary to emphasize that adequate medical management, including elimination of infection, should be employed postoperatively when either primary or secondary ulcer is present.

REVIEW OF CASES REPORTED IN THE LITERATURE

Our review of the literature disclosed four interesting facts. The first is that relatively few exhaustive studies of this subject have been made. The most notable ones in English are those made by Paterson,[65] Massie,[56] and Singer and Meyer.[81] The second interesting fact is that only 93 authenticated case reports of acute perforation of gastrojejunal ulcer could be found. Singer and Meyer[81] state that the publication of Steden's[84] article in 1931 brought the total number of cases reported in the German literature to 20. In the general literature on gastrojejunal ulcer there are a number of casual references to cases of acute perforation into the free peritoneal cavity. Except from the standpoint of numbers these cases have little significance because they cannot in any way be considered as case reports useful for study. For example, Wright[102] mentioned that 51 cases had been reported to him by the Fellows of the Association of Surgeons. Sokolov[82] made a similar collection of 12 cases from among Russian surgeons. Hurst and Stewart[37] referred to eight cases and Graham and Lewis[26] to one case. The third interesting fact is that a number of cases reported as acute in reality have been subacute peforations or perforations into the colon or through the anterior abdominal wall. Singer and Meyer[81] credit Fromme,[24] Florcken and Steden,[22] McKelvey,[58] Recht[71] and Urrutia[94] with a total of ten cases whereas we accept only four of them as cases of acute perforation into the free peritoneal cavity. Moreover, personal communications indicate that through error Lewisohn[51, 52] mentioned one case and that Storey[88, 89] included a case of subacute perforation and a case of primary perforation of the jejunum in his incidental report of five cases of acute perforation of gastrojejunal ulcer. The fourth point of interest is that there is a good

deal of confusion regarding certain cases. As an illustration, Mikulicz is credited as having reported five cases. As early as 1909, Paterson[65, 66] classified three of these as doubtful and our study of the literature has disclosed that neither of the other two is a typical acute perforation of secondary ulceration at the gastrojejunal anastomosis. In 1899 and 1900, Kausch[44] reported from Mikulicz clinic a case of chronic perforating gastrojejunal ulcer which ultimately penetrated the anterior abdominal wall. In the Handbook of Surgery in 1903, Mikulicz and Kausch[60] made a parenthetic reference to a case wherein perforation of an acute gastrojejunal ulcer occurred in an infant soon after operation for hypertrophic stenosis of the pyloris. Freund,[23] in 1903, reported the same case from the standpoint of hypertrophic stenosis without reference to the gastrojejunal ulcer. Mikulicz,[59] also in 1903, reported briefly the same case to the American Surgical Association. Such instances of multiple reports of the same case have served to confuse rather than clarify the situation. In view of the foregoing facts and the fact that authenticated cases of acute perforation of gastrojejunal ulcer now number over 100, it seemed promising to combine these cases for a comprehensive study of a problem which is clinically distinct from that presented by primary peptic ulcer in any of its complicated forms or by gastrojejunal ulcer in any of the other forms in which it is seen to exist.

This study is made on a total of 103 cases, 93 of which were collected from the original case reports in the literature. It is our privilege to add ten new cases which are reported here for the first time. The latter group is composed of five cases of our own and five which were generously contributed to this study. For two of the latter we are indebted to Hinton[34] and for three to Storey[89] (Table VI).

Age and Sex.—Among the 92 cases in which the age was recorded there were cases in every decade of life from the first through the seventh. The single case in the first decade was in a child of five months. In the seventh decade there were two cases. Thirty-six per cent of the cases occurred in the fourth decade and 49 per cent in the third and fifth decades combined. Among the 99 cases in which the sex was recorded, 90 per cent occurred in males. Acute perforation of peptic ulcer therefore is predominantly a disease of the male sex and of the ages from 21 to 50.

Primary Pathology.—The primary pathologic condition for which gastrojejunostomy was performed was recorded in 89 cases (Table I). Duodenal ulcer was present in 43 per cent, gastric ulcer in 30 per cent. Pyloric stenosis was recorded as the primary pathology is 20 per cent of the cases. Carcinoma of the stomach, gastroptosis, gastric erosions, congenital hypertrophic stenosis of the pylorus, gastric and duodenal perforations and "no pathology found," each were reported in one case. It is probable that peptic ulcer was present as the cause of stenosis in many of the cases in the pyloric stenosis group.

TABLE I

PRIMARY PATHOLOGY

	Cases	
	Number	Per Cent
Duodenal ulcer	38	42.6
Gastric ulcer	27	30.3
Pyloric stenosis	18	20.2
Carcinoma of stomach	1	1.1
Gastroptosis	1	1.1
Gastric erosions	1	1.1
Congenital hypertrophic stenosis of pylorus	1	1.1
Gastric and duodenal perforations	1	1.1
No pathology found	1	1.1
Totals	89	99.7

COMPLICATIONS OF PRIMARY PATHOLOGY

	Cases	
	Number	Per Cent
Acute perforation of peptic ulcer	24	27
Peptic ulcer with obstruction	20	22
Peptic ulcer with hemorrhage	11	12
Peptic ulcer with chronic perforation of pancreas	2	2
Totals	57	63

TABLE I—Showing the uncomplicated and complicated primary pathologic conditions for which gastrojejunostomy was performed, to be followed later by acute perforation of a gastrojejunal ulcer. Peptic (gastric and duodenal) ulcer constituted the primary pathology in 72.9 per cent of cases. Acute perforation was the most common complication of peptic ulcer to be followed by acute perforation of gastrojejunal ulcer.

One of the four common complications of peptic ulcer was present in 63 per cent of the 89 cases in which primary pathology was recorded (Table I). Acute perforation of peptic ulcer was present in 27 per cent, peptic ulcer with obstruction in 22 per cent, hemorrhage in 12 per cent, and chronic perforation into the pancreas in 2 per cent of cases. It should be noted that the most common complication of primary peptic ulcer was acute perforation. Obustruction (exclusive of pyloric stenosis) was the next most common complication.

ORIGINAL OPERATION.—The form of the original operation was recorded in all of the 103 cases (Table II). Among these gastrojejunostomy was used in 92 cases, the posterior form in 45 and the anterior in 40 cases. Pyloric resection was utilized in 11 cases with the Billroth II and Pólya types each having been applied in four instances. Operative procedures used in combination with the above in 24 cases were entero-anastomoses, which were used in 16 cases, and pyloric exclusion utilized in five cases,

excision of ulcer and the Roux en Y operation which were used in one and two cases respectively. In making the anastomoses non-absorbable sutures were employed in eight and the Murphy button in four cases.

TABLE II

ORIGINAL OPERATION

	Cases
Gastrojejunostomy	
Posterior	45
Anterior	40
Type not stated	7
Total	92
Pyloric resection	
Billroth II	4
Pólya	4
Hofmeister	1
Type not stated	2
Total	11
Combinations with the above	
Entero-anastomosis	16
Pyloric exclusion	5
Roux en Y	2
Excision of ulcer	1
Total	24
Method of making anastomosis	
Non-absorbable suture	8
Murphy button	4

TABLE II. Showing the types of original operation which were followed by gastrojejunal ulcer complicated by acute perforation. Gastrojejunostomy was employed in 92 cases and pyloric resection in 11 cases. Other operative procedures combined with these were used in 24 cases. In making the anastomoses non-absorbable sutures were used in eight cases and the Murphy button in four cases.

Interval between Gastrojejunostomy and Acute Perforation.—The intervals between the performance of gastrojejunostomy and the occurrence of acute perforation of gastrojejunal ulcer were recorded in 101 cases. The shortest interval during which acute perforation occurred was five days and the longest 18 years. In 13 cases (13 per cent) perforation occurred within three months after operation. In 59 cases (58 per cent) it occurred within two years and in 85 cases (84 per cent) within five years. In only 16 cases (16 per cent) did perforation occur as late as from five to 18 years.

Pathology of Acute Perforation of Gastrojejunal Ulcer.—In the material collected for this study there are 120 acute perforations recorded in the 103 cases. Numerically, therefore, the perforations fall into two groups, one group representing multiple and the other single perforations.

Multiple Perforations.—The multiple perforations, likewise, fall into two groups. One subgroup consists of multiple simultaneous perforations and the other of multiple successive perforations (Table III). In three cases multiple (two) perforations were found to exist simultaneously at operation or autopsy. In one case the site of the perforations was not stated but in each of the other two cases the perforations were situated at the anastomosis and in the jejunum.

TABLE III

MULTIPLE ACUTE PERFORATIONS

Multiple (2) Simultaneous Perforations

Author	Cases	Site
Key	1	Not stated
Massie	1	Anastomosis and efferent limb
Charbonnel and Leuret	1	Anastomosis and jejunum
Total	3	

Multiple Successive Perforations

Author	Cases	Site		Total Acute Perforations
		Peptic Ulcer (Gastric or duodenal)	Gastrojejunal Ulcer	
Battle	1	1	3	4
Maylard	1	1	2	3
Henry	1	1	4	5
Steinberg	1	1	2	3
Riess	1	0	2	2
Robinson	1	0	2	2
Nixon and Lowry	1	0	3	3
Woodside	1	0	2	2
Dineen	1	0	2	2
Hinton and Church	1	0	2	2
Schilling	1	0	2	2
Totals	11	4	26	30

TABLE III.—Tabulates the three cases of multiple simultaneous and 11 cases of multiple, successive, acute perforations of gastrojejunal ulcer.

In 11 cases multiple (from two to five) perforations were found to have occurred at different times. In four of these cases acute perforation of peptic (gastric or duodenal) ulcer preceded acute perforation of gastro-

jejunal ulcer. In this group of 11 cases acute perforation of gastrojejunal ulcer occurred twice in eight cases, three times in two cases, and four times in one case. There were four acute perforations of peptic ulcer which, combined with 26 acute perforations of gastrojejunal ulcer, comprise a total of 30 acute perforations of ulcer in the 11 patients. Acute perforation of peptic ulcer followed by single perforation of gastrojejunal ulcer has been discussed under primary pathology.

Single Perforations.—Single perforation of gastrojejunal ulcer occurred in 89 cases. The important pathologic features in these cases are included in the following section.

Site of Perforation.—In the 120 acute perforations of gastrojejunal ulcer in this study the site was recorded in 96 instances (Table IV). The perforation was in the stomach three times, at the anastomosis in 19 instances, and in the jejunum 74 times. Jeunal perforation occurred in the afferent limb ten times, opposite the anastomosis 19 times and in the efferent limb 45 times.

TABLE IV

SITE OF PERFORATION

	Cases	
	Number	Per Cent
Stomach	3	3.1
At anastomosis	19	19.7
Jejunum		
Afferent limb	10	10.4
Opposite anastomosis	19	19.7
Efferent limb	45	46.8
Totals	96	99.7

ASSOCIATED PATHOLOGY

	Cases
Multiple ulcers	17
Hemorrhage	14
Gastrojejunocolic fistula	6
Gastric ulcer	2
Abscess	2
Obstruction	
At anastomosis	2
At pyloris	1
Total	44

TABLE IV.—Showing the site of perforation of and the pathology associated with acute perforation of gastrojejunal ulcer.

TABLE V

TREATMENT

Primary Operation	Deaths			Secondary Operation Required	
Operation	No. of Cases	Cases	Per Cent Mortality	No of Cases	Operation
Not performed	22	2	90.9	2	Drainage of abscesses
Suture					Disconnection of gastrojejunostomy
Simple suture	51	9	17.6	4	Gastroduodenostomy
With excision of ulcer	6	0	0.0		Pylorectomy Roux
					Pylorectomy Pólya
With entero-anastomosis	5	0	0.0	1	Pyloric resection
With jejunostomy	1	1	100.0		
Totals	63	10	15.8		
Disconnection of gastrojejunostomy	4	0	0.0	1	Posterior gastrojejunostomy
Gastrojejunostomy					
Type not stated	4	1	25.0		
Anterior	2	0	0.0	1	Disconnection of gastrojejunostomy
With entero-anastomosis	1	1	100.0		
Totals	7	2	28.5		
Pyloric resection					
Billroth II	4	0*	0.0		
Pólya	3	0	0.0		
Reichl-Roux	3	0	0.0		
Billroth I	1	0	0.0		
Mikulicz-Reichl	1	0	0.0		
Kronlein-Roux	1	0	0.0		
Hoffmeister	1	0	0.0		
Type not stated	3	1	33.3		
Totals	17	1	5.8		
Other operations					
Type not stated	2	?	†		
Appendectomy	1	1	100.00		
Resection of jejunum, anastomosis en Y and pyloric exclusion with ligature	1	0	0.0		
Totals	4	?	?		

* Death or recovery in 1 case not stated

† Death or recovery not stated

TABLE V.—Comparison of the different types of surgical treatment with results in 117 acute perforations of gastrojejunal ulcer. The mortality in the untreated cases reached the high point of 90.9 per cent whereas in simple disconnection of gastrojejunostomy the mortality was 0.0 per cent.

Associated Pathology.—Complicating pathology was associated with acute perforation of gastrojejunal ulcer in 44 cases. Multiple jejunal ulcers were present in 17 cases. In von Eiselsberg's case there were five jejunal ulcers in addition to a gastrojejunocolic fistula. Hemorrhage had complicated gastrojejunal ulcer in 14 cases. In six gastrojejunocolic fistulae were present. Gastric ulcer, intraperitoneal abscess and obstruction of the anastomotic stoma each was present in two cases and pyloric obstruction was present in one case.

Autopsy.—Autopsy was performed in 25 cases.

Treatment.—In the 22 cases in which primary operation was not performed there were 20 deaths representing a mortality of 90.9 per cent. In both of the patients who recovered secondary drainage of intraperitoneal abscesses was required. Simple suture of the perforation was carried out in 51 cases with nine deaths and a mortality of 17.6 per cent. Simple suture was combined with other procedures in 12 cases with one death. In 63 cases wherein simple suture of the perforation was carried out with or without other procedures the total mortality was 15.8 per cent. Disconnection of the gastrojejunostomy restoring the normal sequencial relation of the stomach and intestinte was utilized in four cases with no deaths. Gastrojejunostomy alone or combined with other procedures was used in seven cases with two deaths and a mortality of 28.5 per cent. Pyloric resection with various types of gastro-intestinal anastomosis was applied in 17 cases with only one death, making a mortality of 5.8 per cent. In the ten cases in which the interval between perforation and operation was mentioned, it was recorded as from immediately up to six hours in seven cases, from seven to 12 hours in two cases and on the next day in one case. In the case in which death resulted, the operation was performed 11 hours after perforation. In four cases other operations ranging from appendectomy to resection of the jejunum with a Y anastomosis and pyloric exclusion by ligature were used. As recovery or death was not recorded in two of these information concerning the mortality in this group is not complete (Table V).

Mortality.—Among the 117 acute perforations of gastrojejunal ulcer in this study the outcome (recovery or death) was not recorded in three instances. In the remaining 114 acute perforations there were 34 deaths, representing a gross mortality of 29.8 per cent.

CASE REPORTS

Case 1.—(Toland and Thompson). Male, aged 42. In 1921, onset of epigastric pain relieved by food and soda. Excision of duodenal ulcer, posterior gastrojejunostomy and cholecystostomy in 1924. Pain recurred six months later. In 1927, medical treatment for duodenal ulcer. In 1928, sudden severe pain in the epigastrium later shifting to the suprapubic region and referred to both shoulders. Physical findings consisted of retracted abdomen with generalized tenderness and board-like rigidity, dulness in the flanks but liver dulness was not obliterated. Diagnosis: Perforated duodenal or jejunal ulcer.

Operation three hours after onset of pain disclosed a large quantity of free fluid

in the pelvis and beneath the great omentum. A perforated ulcer 1.5 cm. in diameter was found on anterior wall of jejunum opposite the anastomosis. The scar of a healed ulcer was situated on the anterior wall of stomach 4 cm. proximal to the pylorus. Pylorus patent. Disconnection of gastrojejunostomy, closure of stomach and resection of a portion of ulcer bearing jejunum 12 cm. long with end-to-end anastomosis by suture. Closure of abdomen with drainage.

Pathologic Report.—Specimen of intestine 6 cm. long. Circular opening 14 Mm. in diameter with smooth mucosal edges apparently a gastrojejunostomy opening. Three cm. directly anterior to this opening is a second opening measuring 9 × 12 Mm. extending through the entire wall of the intestine with indurated and undermined mucosal edges typical of a peptic ulcer. Serosa moderately thickened about the opening and covered by a delicate layer of fibrin. No evidence of malignancy. Gross Diagnosis: Perforated jejunal ulcer. Recovery.

Three months after operation patient was readmitted to hospital for observation for recurrent epigastric pain. Fluoroscopic examination disclosed a dilated stomach and tendency for the barium to collect in the dilated duodenal loop. In 1929, patient was readmitted for persistent pain, after having vomited one quart of coffee ground material, followed by two copious tarry defecations. Red blood corpuscles, 1,480,000. Night retention of from 250 to 650 cc. Medical treatment for two months including transfusion of blood.

Second Operation.—Eighteen months after acute perforation of gastrojejunal ulcer: Many adhesions. Firm mass at pylorus. Head of pancreas hard and enlarged. Posterior no-loop gastrojejunostomy with sutures. General condition of patient too poor to risk resection of tumor at pylorus. Recovered. Readmitted to hospital two months postoperative because of persistence of pain in lower abdomen. Improved under medical treatment. When last examined in October, 1930, patient still complained of pain in the epigastrium. Medical treatment was advised.

Case 2.—(Toland and Thompson). Male, aged 36. In 1917, onset of periodic gnawing pain in the epigastrium appearing two to three hours after meals and relieved by vomiting. Diagnosis: Peptic ulcer. Medical treatment with relief for six months. In 1928, while eating, experienced severe abdominal pain.

Operation six hours after onset of pain disclosed a perforated duodenal ulcer. Sutured. In 1929, posterior gastrojejunostomy with partial relief of symptoms. In 1934, sudden attack of dizziness and vomiting of bright red blood, melena. Medical treatment. Three months later, one and one-half hours after a meal of soft food, patient experienced a sudden severe epigastric pain. Generalized abdominal rigidity was found on physical examination. Diagnosis: Perforated peptic ulcer.

Second operation three and one-half hours after onset of pain disclosed many adhesions but no evidence of peritonitis anterior to the great omentum. Entire region beneath the transverse colon filled with milky fluid. Multiple lesions in the omentum resembling fat necrosis. Perforation of anterior wall of jejunum directly below anastomosis 2 Mm. in diameter. Suture. Closure with drainage. Recovery. On follow-up five months postoperative, patient found to experience ulcer pain at intervals. Diagnosis: Marginal or jejunal ulcer.

Case 3.—(Toland and Thompson). Male, aged 65. In 1924, onset of pain following meals, relieved by soda. In 1929, vomiting of practically all food except fluids. Diagnosis: Peptic ulcer with obstruction. Posterior no-loop gastrojejunostomy for duodenal ulcer with obstruction. In 1934 after severe agonizing epigastric pain readmitted to the hospital where a diagnosis of perforated peptic ulcer was made. The patient stated that he had been free from symptoms until three weeks prior to this admission.

Operation disclosed perforated ulcer of afferent limb of jejunum. Mass in first portion of duodenum had disappeared. Suture and entero-anastomosis. Recovery.

Case 4.—(Toland and Thompson). Male, aged 34. In 1929, gnawing epigastric pain after meals relieved by food, of three weeks' duration when patient experienced a

sudden sharp stabbing epigastric pain. Examination disclosed moist cold skin and rigid abdomen. Diagnosis: Perforated peptic ulcer.

Operation disclosed ulcer anterior wall of pylorus with perforation 1 cm. in diameter. Suture and posterior gastrojejunostomy. Relief until 1934 when there was a recurrence of severe abdominal pain. Abdomen board-like and tender throughout. Diagnosis: Perforated peptic ulcer. Operation: Perforated ulcer at the margin of the gastrojejunostomy opening. Clear fluid in peritoneal cavity. Closure of perforation with sutures. Drainage of peritoneal cavity. On sixth day postoperative evisceration through a small opening in the incision. Secondary closure. Patient died on sixteenth day postoperative. Autopsy: Generalized peritonitis. Ulcer in greater curvature of stomach communicating with jejunum. Bilateral fibrinous pleuritis.

Case 5.—(Toland and Thompson). Male, aged 37. Gastrojejunostomy eight years ago for gastric ulcer. Recent examination for recurrence of symptoms at the Mayo Clinic resulted in a diagnosis of marginal ulcer and reactivation of duodenal ulcer. In September, 1935, patient experienced a sudden pain in the right upper quadrant of the abdomen. Examination disclosed a flat, rigid, tender anterior abdominal wall and obliteration of liver dulness. Diagnosis: Acute perforation of duodenal ulcer.

Operation disclosed an active duodenal ulcer, which was not ruptured, and an acute perforation of an ulcer in the efferent limb of the jejunum. Gentle manipulation of the jejunum caused a separation throughout half of the circumference of the anastomosis. Disconnection of gastrojejunostomy with suture of gastric and jejunal openings and restoration of normal anatomy. Recovery.

Case 6.—(Hinton). Male, aged 45. Posterior gastrojejunostomy for duodenal ulcer in 1920. In 1933, acute perforation of ulcer of efferent limb of jejunum. Simple suture. Recovery. Recurrence of pain. Further surgery advised.

Case 7.—(Hinton). Male, aged 55. Posterior gastrojejunostomy in 1929. On February 25, 1936, acute perforation of gastrojejunal ulcer situated in the efferent limb. Simple suture. Recovery.

Case 8.—(Storey). Male, aged 44. In 1924, posterior gastrojejunostomy for duodenal ulcer with obstruction. Relief for 11 months, when pain in the epigastrium associated with melena appeared. Gastric analysis disclosed hyperacidity and fluoroscopic examination with a barium meal revealed an accessory pocket near the stoma suggestive of ulcer. In 1925, operation disclosed a cavity filled with brown fluid situated between the jejunum and transverse mesocolon into which a perforation of the jejunum emptied. A piece of silk three inches long was removed from the neighborhood of the ulcer. The duodenum was contracted to the diameter of a little finger. Gastrojejunostomy disconnected, openings closed, anterior gastrojejunostomy performed. Recovery.

Case 9.—(Storey). Male, aged 52. In 1923, after having had epigastric pain for 12 months, anterior gastro-enterostomy and entero-anastomosis were performed. In 1928, sudden severe pain in the region of the umbilicus.

Operation disclosed adhesions between the omentum, stomach and abdominal wall and free fluid in the peritoneal cavity. A small perforation in the efferent limb of the jejunum was sutured. Recovery.

Case 10.—(Storey). Male, aged 47. In 1929, posterior gastrojejunostomy for duodenal ulcer with obstruction. In 1932, perforated bleeding jejunal ulcer opposite the stoma closed with sutures. Recovery.

Discussion.—In addition to the contribution of ten new cases of acute perforation of gastrojejunal ulcer to the literature, this study of 103 cases has emphasized a number of significant facts. They will be discussed under three broad headings; namely, etiology, pathology, and treatment.

Etiology.—This study reemphasizes that while acute perforation of gastrojejunal ulcer may occur at any period from infancy to old age, the fact that 85 per cent of cases were found to occur from the third through the fifth

decade indicates that the condition is distinctly a disease of early and middle adult life. By a similarly large percentage of cases the condition also is seen to predominate in males.

An analysis of the data on the primary pathology has brought out two important facts. First, it confirms the prevailing impression that acute perforation of gastrojejunal ulcer occurs most frequently following peptic ulcer. What is more important, however, is that it illustrates how perforation of gastrojejunal ulcer may occur not only after malignancy of the stomach but also as an untoward sequela to gastrojejunostomy when performed in such benign conditions as gastric erosions, gastroptosis or even in the absence of pathology. These points serve to emphasize further the inadvisability of application of this operation in functional conditions or when pathology is not demonstrable or is indefinite.

The second important fact is that pyloric stenosis was recorded as being the primary pathology in 18 cases. It will be noted that most of these cases were reported by early writers. One of us (H.L.T.[41]), in collaboration with Judd, has shown that while primary pyloric stenosis exists, its occurrence among adults is rare. While it is probable, therefore, that many if not all of the cases in this group were in reality pyloric or duodenal ulcer with secondary stenosis, the absence from the case reports of data which would enable us to distinguish the two forms made it necessary for us to recognize two groups. Had we combined the groups there would have been 38 cases or as high as 36.8 per cent of the entire series wherein acute perforation of gastrojejunal ulcer followed pyloric obstruction. This is of special interest in view of Wilkie's[96] recent statement that gastrojejunostomy should be performed only in cases of pyloric obstruction.

Analysis of the data on complications of the primary pathology is also significant. It is seen that complications were present in 63 per cent of the cases in which gastrojejunostomy was used. Acute perforation of peptic ulcer outnumbers peptic ulcer with obstruction by 5 per cent and becomes therefore the most common complication to be followed by acute perforation of gastrojejunal ulcer. By carrying the analysis to the point of distinguishing between the tendencies manifest by peptic ulcer we find that the liability to reactivation, as indicated by acute and chronic perforation and by erosion of blood vessels, outstrips the tendency to inactivity as shown by scarring and obstruction in a case ratio of 37 to 20.

The secondary nature and occurrence of gastrojejunal ulcer brings our consideration of the types of original operation under the heading of etiology. As gastrojejunostomy *per se* is an essential prerequisite to the formation of gastrojejunal ulcer, it is necessary for us to consider only the various types of this operation and the effects of the operations which were used in combination with it.

The comparative frequency of acute perforation of gastrojejunal ulcer with respect to anterior or posterior gastrojejunostomy is striking. During the early history of gastrojejunal ulcer when anterior gastrojejunostomy was used more often than at present, the impression prevailed among students of

TABLE VI

SUMMARY OF REPORTED CASES OF GASTROJEJUNAL ULCER*

No.	Reported by Author	Year	Age in Years	Sex	Original Pathology	Original Operation	Interval	Pathology of Gastrojejunal Ulcer	Operation for Gastrojejunal Ulcer	Result	Remarks
1	Braun[6]	1899	25	M.	Gastric ulcer, obstruction	Post. gastrojejunostomy	11 mos.	Jejunum, efferent	None	Death	
2	Hahn [30]	1899	—	—	Pyloric stenosis	Ant. gastrojejunostomy	1 yr.	Jejunum, afferent	None	Death	
3	Korte[46]	1900	30	M.	Gastric ulcer, obstruction	Ant. gastrojejunostomy	3 yrs.	Jejunum, efferent	Appendectomy	Death	
4	Steinthal[87]	1900	44	M.	Chronic gastritis, hemorrhage	Post. gastrojejunostomy, Murphy button	10 da.	Jejunum, efferent	None	Death	
5	Goepel[25]	1902	—	M.	Pyloric stenosis	Ant. gastrojejunostomy	13 mos.	—	None	Death	See also Tiegel
6	Goepel[25]	1902	—	M.	Pyloric stenosis	Ant. gastrojejunostomy	4 mos.	—	Suture	Recovery	First case operated upon
7	Mikulicz[59]	1903	5 mos.	F.	Congenital hypertrophic stenosis, obstruction	Ant. gastrojejunostomy, entero-anastomosis	82 da.	Jejunum, afferent. Multiple ulcers. Hemorrhage	None	Death	
8	Brentano[7]	1903	26	F.	Pyloric stenosis	Ant. gastrojejunostomy, entero-anastomosis	1 yr.	Anastomosis	New gastro-jejunostomy	Recovery	
9	Cackovic[11]	1903	30	M.	Pyloric stenosis, obstruction	Post. gastrojejunostomy	5 da.	Jejunum, efferent Multiple ulcers	None	Death	
10	Tiegel[92]	1904	34	M.	——	Ant. gastrojejunostomy	9 mos.	—	Suture	Recovery	
11	Dudgeon and Sargent[17]	1905	30	M.	Gastric ulcer	——	22 mos.	—	Suture	Recovery	
12	Battle[2]	1906	30	M.	Gastric ulcer, obstruction, hemorrhage	Ant. gastrojejunostomy, Murphy button	22 mos.	—	Suture	Recovery	
13	I† Battle[2]	1906	37	F.	Gastric ulcer, obstruction, acute perforation	Ant. gastrojejunostomy	13 mos.	Jejunum, efferent	Suture	Recovery	
	II Battle[2]	1906		F.	Gastric ulcer, obstruction, acute perforation	Ant. gastrojejunostomy	23 mos.	Anastomosis	Suture	Recovery	
	III Romanis[75]	1918		F.	Gastric ulcer, obstruction, acute perforation	Ant. gastrojejunostomy	13 yrs.	Jejunum, efferent	Suture	Recovery	
14	Delaloye[14]	1906	41	M.	Duodenal ulcer, obstruction	Ant. gastrojejunostomy	6 yrs.	Anastomosis	None	Death	
15	Graser[27]	1906	—	F.	Gastric ulcer, obstruction	Post. gastrojejunostomy, Murphy button	4 yrs.	Jejunum, efferent, hemorrhage	None	Death	
16	Eiselsberg[20]	1906	—	M.	Gastric ulcer	Billroth II	1 yr.	Stomach	None	Death	
17	Paterson[64, 65, 66]	1906	48	M.	Pyloric stenosis	Post. gastrojejunostomy	2 yrs.	Jejunum, efferent	None	Death	
18	Paterson[64, 65, 66]	1906	51	M.	Pyloric stenosis	Ant. gastrojejunostomy	2½ yrs.	Jejunum, opposite anastomosis	None	Death	
19	Key[48]	1907	45	F.	Gastric ulcer, obstruction, hemorrhage, perforation into pancreas	Ant. gastrojejunostomy Y	7 yrs.	Anastomosis	Excision of ulcer, enlargement of anastomosis, suture	Recovery	
20	Key[45]	1907	25	F.	Carcinoma stomach	Billroth II	10 da.	Multiple ulcers, 2 simultaneous perforations	None	Death	

* In the compilation of the data in this table of collected cases the original case report was consulted in every instance.

† The Roman numeral indicates the number of each perforation as it occurred in cases in which multiple successive perforations took place.

21	Edington[18]	1907	46	M.	Duodenal ulcer, obstruction	Ant. gastrojejunostomy	7 yrs.	Jejunum, efferent	Suture	Death	
22	Hamann[31]	1907	48	M.	Gastric erosions	Ant. gastrojejunostomy, Murphy button, Entero-anastomosis	26 da.	Jejunum, efferent	None	Death	
23	Jainu[38]	1907	—	M.	—— Hemorrhage	Post. gastrojejunostomy	11 mos.	Jejunum, opposite anastomosis	None	Death	
24	Jensen[39]	1908	54	M.	Pyloric stenosis	Post. gastrojejunostomy	13 mos.	Jejunum, efferent	None	Death	
25	Lion and Moreau[55]	1909	43	M.	Pyloric stenosis	Post. gastrojejunostomy	8 yrs.	Jejunum, afferent, hemorrhage, jejunocolic fistula	None	Death	
26	Eiselsberg[21]	1910	40	M.	Gastric ulcer	Post. gastrojejunostomy	2 mos.	Jejunum, opposite anastomosis, multiple (5) ulcers, jejunocolic fistula	New gastro-jejunostomy, entero-anastomosis	Death	
27	I Maylard[57]	1910	51	F.	Gastric ulcer, acute perforation	Ant. gastrojejunostomy	8 mos.	Jejunum, efferent	Suture	Recovery	
	II Maylard[57]	1910	51	F.	Gastric ulcer, acute perforation	Ant. gastrojejunostomy	3 yrs.	Jejunum, efferent	Suture	Recovery	
28	Battle[3]	1910	39	M.	Gastric ulcer, obstruction, acute perforation	Ant. gastrojejunostomy	2 yrs.	Anastomosis	Suture	Recovery	
29	Petren[67]	1911	37	M.	Gastric ulcer, acute perforation	Post. gastrojejunostomy, entero-anastomosis	9 mos.	Anastomosis	Excision of ulcer, suture	Recovery	
30	Petren[68]	1911	18	M.	Gastric ulcer, obstruction, hemorrhage	Post. gastrojejunostomy	5 da.	Jejunum, afferent, multiple ulcers	None	Death	
31	Hitzrot[36]	1911	26	M.	Gastric ulcer, obstruction	Ant. gastrojejunostomy, Roux Y	7 yrs.	Jejunum, efferent	Suture	Recovery	
32	Moynihan[61]	1912	29	M.	Pyloric stenosis	Post. gastrojejunostomy	7 da.	Jejunum, opposite anastomosis	Suture	Death	
33	Rowlands[76, 77]	1912	30	M.	Pyloric stenosis	Ant. gastrojejunostomy	5 yrs.	Jejunum, efferent	Suture	Recovery	Secondary operation: Disconnection
34	Lindner[54]	1912	50	F.	——	——	—	——	None	Death	Secondary operation: Gastrojejunostomy Died 2 days post-operative of acute perforation at first gastrojejunostomy
35	Lindner[54]	1912	—	M.	——	——	6 yrs.	Anastomosis	Suture	Recovery	
36	Hartmann[32]	1913	41	M.	Pyloric stenosis	Post. gastrojejunostomy	6 yrs.	Anastomosis	Suture	Recovery	
37	Schmilinsky[79]	1918	—	—	Duodenal ulcer	Post. gastrojejunostomy, pyloric exclusion	22 mos.	——	Suture	Recovery	
38	Schmilinsky[79]	1918	—	—	Duodenal ulcer	Post. gastrojejunostomy, pyloric exclusion	16 mos.	Jejunum, efferent	Suture	Death	
39	Haberer[28, 29]	1918	40	M.	Duodenal ulcer	Post. gastrojejunostomy, pyloric exclusion	58 da.	—— Multiple ulcers, (4) colic fistula	Suture	Death	
40	Wright[101]	1919	39	M.	Duodenal ulcer, obstruction	Ant. gastrojejunostomy	19 mos.	Jejunum, efferent	Suture	Recovery	

COLLECTED CASES—*Continued*

No.	Reported by Author	Year	Age in Years	Sex	Original Pathology	Original Operation	Interval	Pathology of Gastrojejunal Ulcer	Operation for Gastrojejunal Ulcer	Result	Remarks
41	Roberts[73]	1920	34	M.	Pyloric stenosis, acute perforation	Post. gastrojejunostomy	2½ mos.	Jejunum, afferent	Suture	Recovery	
42	Polya[69]	1920	36	M.	Duodenal ulcer	Post. gastrojejunostomy, pyloric exclusion	10 mos.	Jejunum, opposite anastomosis, multiple (4) ulcers	Suture	Death	
43	I Henry[33]	1921	36	M.	Gastric ulcer, acute perforation	Ant. gastrojejunostomy	5 mos.	Jejunum, opposite anastomosis	Suture	Recovery	
	II Henry[33]	1921	—	M.	Gastric ulcer, acute perforation	Ant. gastrojejunostomy	2½ yrs.	Jejunum, efferent	Suture	Recovery	
	III Henry[33]	1921	—	M.	Gastric ulcer, acute perforation	Ant. gastrojejunostomy	4½ yrs.	——	Suture	Recovery	
	IV Henry[33]	1921	—	M.	Gastric ulcer, acute perforation	Ant. gastrojejunostomy	6 yrs.	Anastomosis	Excision of ulcer, suture	Recovery	
44	Urrutia[94]	1921	35	M.	Duodenal ulcer, acute	Post. gastrojejunostomy	14 mos.	Jejunum, efferent	Suture	Recovery	Secondary operation: Roux resection
45	Haberer[29]	1922	34	—	Gastric ulcer	Post. gastrojejunostomy	4 yrs.	——	Suture, jejunostomy	Death	
46	Brutt[9]	1922	18	M.	Pyloric stenosis	Ant. gastrojejunostomy	3 yrs.	——	New gastro-jejunostomy	Recovery	
47	Brutt[9]	1922	40	M.	Pyloric stenosis	Ant. gastrojejunostomy	6 yrs.	Jejunum, efferent, hemorrhage	Suture	Death	
48	McKelvey[58]	1922	44	M.	Gastric ulcer	Ant. gastrojejunostomy	18 yrs.	Anastomosis	Disconnection	Recovery	
49	Massie[56]	1924	28	M.	Duodenal ulcer	Ant. gastrojejunostomy	16 mos.	Multiple simultaneous perforations (2): Anastomosis, jejunum, afferent	Suture	Recovery	Secondary operation: Disconnected
50	Bundschuh[8]	1924	49	M.	Gastric ulcer	Ant. gastrojejunostomy, entero-anastomosis	14 yrs.	Jejunum, efferent, multiple ulcers (2): Acute perforation, Jejunocolic fistula	Pyloric resection, Mikulicz-Reichel anastomosis	Recovery	
51	I Steinberg[85]	1924	36	M.	Duodenal ulcer, acute perforation	Post. gastrojejunostomy	—	Jejunum, efferent, multiple ulcers	Suture	Recovery	
	II Steinberg[86]	1936	—	M.	——	——	—	—— Jejunum	Suture	Recovery	
52	Schwarz[80]	1925	—	M.	Gastric ulcer	Ant. gastrojejunostomy	3 yrs.	Jejunum, opposite anastomosis	None	Death	
53	Schwarz[80]	1925	39	M.	Gastric ulcer	Ant. gastrojejunostomy	1 yr.	Anastomosis, multiple ulcers	Pyloric resection, Reichel-Roux anastomosis	Recovery	
54	Schwarz[80]	1925	33	F.	Duodenal ulcer	Ant. gastrojejunostomy	1½ yrs.	Jejunum, efferent	Pyloric resection, Reichel-Roux anastomosis	Recovery	
55	I Riess[72]	1925	27	M.	Gastric ulcer, perforation into pancreas	Ant. gastrojejunostomy	86 da.	Jejunum, opposite anastomosis	Excision of ulcer, suture	Recovery	

	II Reiss[72]	1925	—	M.	——	——	9 mos.	Jejunum, afferent, multiple ulcers	Pyloric resection, Billroth II. anastomosis	Recovery	
56	Charbonnel and Leuret[12]	1925	60	M.	Gastric ulcer, obstruction	Post. gastrojejunostomy	3 yrs.	Multiple (2) simultaneous perforations: Anastomosis, jejunum, efferent	Suture	Death	
57	Zeno[103]	1926	23	M.	Duodenal ulcer, acute perforation	Post. gastrojejunostomy	5 mos.	Anastomosis, multiple ulcers	Ant. gastro-jejunostomy	Recovery	Secondary operation: Disconnection both gastrojejunostomies
58	Ashcroft[1]	1925	38	M.	Duodenal ulcer, acute perforation	Post. gastrojejunostomy	19 mos.	Jejunum, efferent	Suture, entero-anastomosis	Recovery	Secondary operation: Pyloric resection
59	Ashcroft[1]	1926	37	M.	Duodenal ulcer	Post. gastrojejunostomy	9 mos.	Jejunum, efferent	Excision of ulcer, suture	Recovery	
60	Winkelbauer[98]	1926	22	M.	Duodenal ulcer, hemorrhage, acute perforation	Ant. gastrojejunostomy	10 mos.	Jejunum, opposite anastomosis	Pyloric resection, Pólya anastomosis	Recovery	
61	Kunz[47]	1926	34	M.	Pyloric stenosis	Ant. gastrojejunostomy, entero-anastomosis	16 mos.	——	Pyloric resection	Recovery	
62	Kunz[47]	1926	31	M.	No ulcer found	Ant. gastrojejunostomy	16 mos.	Jejunum, opposite anastomosis	Pyloric resection, Reichel-Roux anastomosis	Recovery	
63	Delor y Castro[15]	1926	49	M.	Duodenal ulcer	—— Gastrojejunostomy	2 yrs.	Anastomosis	Pyloric resection	Death	
64	Spath[83]	1927	33	M.	Duodenal ulcer	Ant. gastrojejunostomy, entero anastomosis	7 mos.	Jejunum, opposite anastomosis	Pyloric resection, Billroth I	Recovery	
65	Jura[42]	1927	37	M.	Duodenal ulcer, hemorrhage	Post. gastrojejunostomy	6 da.	Jejunum, efferent	None	Death	
66	Brutt[10]	1927	40	M.	—— Acute perforation	Billroth II	1 yr.	—— Multiple ulcers.	Suture	Death	
67	Leveuf[49]	1927	47	M.	——	Post. gastrojejunostomy	5 yrs.	Jejunum, efferent	Resection of jejunum, anastomosis Y, pyloric exclusion with ligature	Recovery	
68	Porzelt[70]	1928	31	M.	Duodenal ulcer, acute perforation	Ant. gastrojejunostomy, entero-anastomosis	7½ mos.	Jejunum, opposite anastomosis	Pyloric resection, Kronlein-Roux anastomosis	Recovery	
69	I Robinson[74]	1928	26	M.	Duodenal ulcer	Billroth II	7 mos.	——	Suture	Recovery	
	II Robinson[74]	1928	—	M.	——	——	1 yr.	Jejunum, efferent	Suture	Recovery	
70	Ehrlich[19]	1928	24	M.	Gastric ulcer, doudenal ulcer, acute perforations (2) simultaneous	Post. gastrojejunostomy	4½ mos.	Jejunum, efferent	Suture	Recovery	
71	I Nixon and Lowry[62]	1928	36	M.	Gastric ulcer, hemorrhage	Post. gastrojejunostomy	15 mos.	Stomach	Suture	Recovery	
	II Nixon and Lowry[62]	1928	—	M.	——	——	5 yrs.	Jejunum, efferent	Suture	Recovery	
	III Nixon and Lowry[62]	1928	—	M.	——	——	6 yrs.	Stomach, multiple ulcers	Suture	Recovery	Secondary operation: Pólya resection

COLLECTED CASES—*Continued*

No.	Reported by Author	Year	Age in Years	Sex	Original Pathology	Original Operation	Interval	Pathology of Gastrojejunal Ulcer	Operation for Gastrojejunal Ulcer	Result	Remarks
72	Just[43]	1929	29	M.	Duodenal ulcer, acute perforation	Ant. gastrojejunostomy, entero-anastomosis	10 mos.	Jejunum, opposite anastomosis	Pyloric resection, Pólya anastomosis	Recovery	
73	Fromme[24]	1929	41	M.	Duodenal ulcer, acute perforation	Ant. gastrojejunostomy, entero-anastomosis	9 mos.	Jejunum, opposite anastomosis	Pyloric resection, Billroth II anastomosis	Recovery	
74	Fromme[24]	1929	32	M.	Duodenal ulcer, acute perforation	Ant. gastrojejunostomy, entero-anastomosis	77 da.	——	Pyloric resection, Billroth II anastomosis	—	
75	I Woodside[100]	1931	16	M.	Duodenal ulcer	Post. gastrojejunostomy	4 yrs.	Jejunum, efferent	Suture	Recovery	
	II Woodside[100]	1931	—	M.	——	——	6 yrs.	Jejunum, afferent	Suture	Recovery	
76	Steden[84]	1931	24	M.	Gastroptosis	Ant. gastrojejunostomy, entero-anastomosis	7 mos.	Jejunum, efferent	Pyloric resection	Recovery	
77	Steden[84]	1931	21	M.	Pyloric stenosis, acute perforation	Ant. gastrojejunostomy, entero-anastomosis	8 yrs.	Jejunum, afferent pyloric stenosis	Pyloric resection, Billroth II anastomosis	Recovery	
78	Steden[84]	1931	30	M.	Gastric ulcer, acute perforation	Pyloric resection	4 yrs.	Jejunum, opposite anastomosis, multiple ulcers, jejunocolic fistula	Pyloric resection, Pólya anastomosis	Recovery	
79	Davenport[13]	1931	5	M.	Duodenal ulcer, hemorrhage	Post. gastrojejunostomy	5 yrs.	——	Suture	Recovery	
80	Brantner and Tonnis[5]	1932	—	M.	Duodenal ulcer, acute perforations (2) consecutive	Pyloric resection	4 mos.	Anastomosis	Suture	Recovery	
81	Bertrand and Etienne-Martin[4]	1933	61	M.	Gastric ulcer, acute perforation	Post. gastrojejunostomy	3½ yrs.	Jejunum, opposite anastomosis	Suture, entero-anastomosis	Recovery	
82	I Dineen[16]	1934	27	M.	Duodenal ulcer, hemorrhage	Pólya resection	4 yrs.	Jejunum, efferent, hemorrhage	Suture	Recovery	
	II Dineen[1]	1934	—	M.	——	——	4½ yrs.	Jejunum, efferent	Suture, entero-anastomosis	Recovery	
83	Singer and Meyer[81]	1934	42	F.	Pyloric stenosis	——	1 yr.	Jejunum, efferent	Suture	Recovery	
84	Singer and Meyer[81]	1934	38	M.	——	Pólya resection	1 yr.	Jejunum, hemorrhage	None	Recovery	Secondary operation: Drainage abscess
85	Singer and Meyer[81]	1934	50	M.	—— Acute perforation	——	3 yrs.	Jejunum, efferent, hemorrhage, gastrocolic fistula	New gastrojejunostomy	Death	
86	Singer and Meyer[81]	1934	49	M.	——	Post. gastrojejunostomy	3 yrs.	Jejunum, stoma obstruction	None	Recovery	Secondary operation: Drainage abscess
87	Singer and Meyer[81]	1934	43	M.	—— Obstruction	——	14 yrs.	Anastomosis	Suture	Recovery	

88	I Hinton and Church[35]	1934	27	M.	Duodenal ulcer, hemorrhage	Pólya resection	4 yrs.	Jejunum, efferent, hemorrhage	Suture	Recovery	
	II Hinton and Church[35]	1934	—	M.	——	——	5 yrs.	Jejunum, efferent	Suture, entero-anastomosis	Recovery	
89	Lewisohn[31]	1934	53	M.	Duodenal ulcer	Hofmeister resection	3 yrs.	Anastomosis, stoma obstruction	Pyloric resection Hofmeister anastomosis	Recovery	
90	I Schilling[78]	1935	31	M.	Duodenal ulcer	Pólya resection, entero-anastomosis	2 yrs.	—— Multiple ulcers	Excision of ulcer, suture, new gastrojejunostomy	Recovery	
	II Schilling[78]	1935	—	M.	——	——	2 yrs.	——	Suture, resection	Recovery	
91	Trevor[93]	1935	33	M.	Duodenal ulcer	Post. gastrojejunostomy	18 mos.	Jejunum, efferent	—	—	
92	Trevor[93]	1935	51	M.	——	Post. gastrojejunostomy	8 yrs.	Jejunum, hemorrhage	—	—	
93	Wilkinson[97]	1935	32	M.	Gastric ulcer	Post. gastrojejunostomy	14 mos.	Jejunum, efferent, hemorrhage	Disconnection	Recovery	
94	Toland and Thompson	1936	42	M.	Duodenal ulcer	Excision of ulcer, post. gastrojejunostomy	4 yrs.	Jejunum, opposite anastomosis	Disconnection	Recovery	Secondary operation: Post. gastrojejunostomy
95	Toland and Thompson	1936	36	M.	Duodenal ulcer, acute perforation	Post. gastrojejunostomy	5 yrs.	Jejunum, opposite anastomosis, hemorrhage	Suture	Recovery	
96	Toland and Thompson	1936	65	M.	Duodenal ulcer, obstruction	Post. gastrojejunostomy	3 yrs.	Jejunum, afferent	Suture, entero-anastomosis	Recovery	
97	Toland and Thompson	1936	34	M.	Gastric ulcer, acute perforation	Post. gastrojejunostomy	5 yrs.	Anastomosis	Suture	Death	
98	Toland and Thompson	1936	37	M.	Duodenal ulcer	Post. gastrojejunostomy	8 yrs.	Jejunum, efferent	Disconnection	Recovery	
99	Hinton	1936	45	M.	Duodenal ulcer	Post. gastrojejunostomy	13 yrs.	Jejunum, efferent	Suture	Recovery	
100	Hinton	1936	55	M.	——	Post. gastrojejunostomy	7 yrs.	Jejunum, efferent	Suture	Recovery	
101	Storey	1936	44	M.	Duodenal ulcer, obstruction	Post. gastrojejunostomy	1 yr.	Jejunum, hemorrhage	New ant. gastrojejunostomy	Recovery	
102	Storey	1936	52	M.	——	Ant. gastrojejunostomy, entero-anastomosis	5 yrs.	Jejunum, efferent	Suture	Recovery	
103	Storey	1936	47	M.	Duodenal ulcer, obstruction	Post. gastrojejunostomy	2½ yrs.	Jejunum, opposite anastomosis, hemorrhage	Suture	Recovery	

this subject that perforating gastrojejunal ulcer was more commonly a sequela to anterior rather than to posterior gastrojejunostomy. Our figures reveal, however, that the condition is even more common after posterior gastrojejunostomy, so that the recorded cases now outnumber the others by 45 to 40.

The occurrence of acute perforation of gastrojejunal ulcer after pyloric resection is of interest from a physiologic standpoint. The impression is prevalent among physicians that pylorectomy reduces the secretion of hydrochloric acid. It has been shown experimentally by one of us (H.L.T[90]) that this depends also on the amount of stomach mucosa resected and on the type and quantity of food consumed. Also of interest from a physiologic standpoint is the large number of acute perforations of gastrojejunal ulcer which occurred after entero-anastomosis. It would appear from these figures that any procedure which shunts the alkaline duodenal juices around the gastrojejunal stoma predisposes strongly to the formation of gastrojejunal ulcer. The use of mechanical devices such as the Murphy button and of non-absorbable sutures appear also to be predisposing factors.

Our findings that 58 per cent of acute perforations of gastrojejunal ulcer occur within two years are in agreement with the recent statements of Judd and Hoerner[40] in this regard.

Pathology.—The pathology of acute perforation of gastrojejunal ulcer especially with respect to multiple perforations is of interest. In view of the fact that multiple gastrojejunal ulcers are relatively common, it is not unusual that a few cases of multiple simultaneous perforations should be encountered.

From the clinical and therapeutic standpoint, one of the most important phases of this study is that of multiple successive acute perforations. The striking tendency of peptic and gastrojejunal ulcers to perforate is amply illustrated by acute perforation of peptic ulcer having preceded acute perforation of gastrojejunal ulcer in 24 cases and by the latter having occurred from two to four times in 11 cases. From this apparent total of 35 cases, four cases must be subtracted as common to both groups, leaving a real total of 31 cases which is 30 per cent of the entire series. When we note that gastrojejunocolic fistula was also associated with acute perforation in six cases, the point is even more striking. This evidence appears to be sufficiently convincing to justify the dictum that gastrojejunostomy should never be performed in unobstructed acute perforation of peptic ulcer. The tendency of peptic and gastrojejunal ulcers to perforation is so strong that in a significant proportion of cases it manifests itself over a period of weeks or years in multiple perforations.

The site of perforation having been found to be more frequent about the artificial stoma, and opposite or distal to it in the jejunum, supports the tissue susceptibility theory of Ochsner[63] and indicates that the corrosive effect of the acid gastric juice plays a highly important rôle in the production and perforation of gastrojejunal ulcer.

Treatment.—The data which we have collected amply illustrate the efficacy

of surgical treatment and the futility of expectant treatment. As against a mortality of 90.9 per cent in the untreated cases, the highest mortality in the treated cases was 28.5 per cent. While the numbers of cases in some of the operation groups are admittedly small, the mortality rates, we believe, are significant. Disconnection of gastrojejunostomy appears to be the safest procedure and probably should be carried out when the patency of the pylorus will permit. Simple suture carrying a mortality of 17.6 per cent and requiring more secondary operations than the other procedures is by virtue of its simplicity applicable to the largest number of cases. This fact alone would tend to make the mortality for this operation appear high. Gastrojejunostomy, which has a mortality in this series of 25 to 28.5 per cent, appears, in view of our other findings, as not only ineffectual but perhaps meddlesome, except in cases wherein pyloric obstruction coexists. The most remarkable finding with respect to treatment of acute perforation of gastrojejunal ulcer was the fact that in 17 cases wherein pyloric resection was carried out there was only one death, representing a mortality of 5.6 per cent.

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REPORT OF COMMITTEE ON ARCHIVES

JOSEPH COLT BLOODGOOD

1867–1935

Joseph Colt Bloodgood was born in Milwaukee, November 1, 1867. He was descended from a family long prominent in legal circles. He was graduated from the University of Wisconsin with the degree of B.S. in 1888, and from the University of Pennsylvania with the degree of M.D. in 1891.

Joseph Colt Bloodgood, M.D.

A year later he joined the Staff of the Johns Hopkins Hospital as Assistant Resident Surgeon. From that time on his promotion was steady through all grades, until at his death he was Adjunct Clinical Professor of Surgery in the Johns Hopkins University and Hospital, and Director of the Garvan Research Laboratory. He was also Chief Surgeon to St. Agnes' Hospital, and Director of the Cancer Research Fund bearing his name.

While Doctor Bloodgood always retained an active interest in general surgery, he early devoted himself to the study of surgical pathology. In later years, cancer research, more especially its cause, early recognition and prevention, attracted his attention. To this end he was an ardent advocate of publicity in all forms, in order to educate the public to seek medical advice early. As a means of stimulating the profession in the prompt recognition of the disease, he was the chief sponsor for a series of meetings of a group of those especially interested in the study of malignant diseases.

Doctor Bloodgood's contributions to scientific medicine have been more especially along the lines just indicated. But early in the history of the Johns Hopkins hospital, Doctor Bloodgood, as his surgical resident, was of material assistance to Professor Halsted in working out various problems in surgical technic, more especially the development of the use of rubber gloves.

Doctor Bloodgood's scientific enthusiasm was unbounded; his energy tireless. While his methods of thought and action did not always conform entirely to orthodox customs, and hence were at times misunderstood by some, yet to the inner circle of his close friends and chosen associates he was ever an interesting personality, an inspiring teacher, a great surgeon, and a loyal friend.

John M. T. Finney.

WILLIAM BRADLEY COLEY

1862–1936

"Ffight on, my men," sayes Sir Andrew Barton,
"I am hurt, but I am not slaine;
I'le lay mee downe and bleed a-while,
And then I'll rise and fight againe!"

How often have we who have known Dr. William B. Coley heard him recite the challenging lines of that old Scottish ballad! In looking back over

William Bradley Coley, M.D.

his life of three-score and 14 years, one is impressed by the fact that it was indeed a series of hard fought battles—battles for what he believed to be the right, not only in the world of science but in affairs of everyday life. His passing on April 16, 1936, has left us with a sense of loss of great leadership.

For one who has been in close association with Doctor Coley for a period

of so many years as I have, it is difficult if not almost impossible to assume the dispassionate rôle of biographer. However, his achievements in the field of surgery as well as in that of cancer research stand out with such Appalachian clearness that one can point to these lofty peaks with little difficulty.

Doctor Coley believed that heredity played the most important part in a man's life; and it would seem to have had a strong influence on his own. He was born in Westport, Connecticut, on January 12, 1862, of an old New England family. His paternal ancestor was Samuel Coley, one of the forty-nine free planters who settled in Milford in 1639. His maternal ancestor was John Wakeman of Benchley, England, a "freeman of the courte of New Haven" (1639) whose son, the Reverend Samuel Wakeman (Harvard, 1655), was one of the first Congregational ministers of Fairfield, Connecticut.

Doctor Coley entered Yale College in the fall of 1880 and was graduated with the degree of Bachelor of Arts in 1884. He taught Latin and Greek at the Bishop Scott Government School in Portland, Oregon, for two years, and then entered the Harvard Medical School in 1886 where he completed the three-year course in two years and was graduated in 1888. As he often remarked, he had taken up the study of medicine and surgery at a most opportune time—when the older surgery with the high mortality from infection was just beginning to be replaced by the newer surgery based on Lister's recognition of the principle of antisepsis. After a strenuous competitive examination, Doctor Coley was appointed an intern on the surgical service of the New York Hospital under Dr. Robert F. Weir and Dr. William T. Bull.

Even at that early date (1888) he showed a keen interest in the subject of sarcoma; and in going over the files of the New York Hospital he came across a case that made a very deep impression upon him. This was a four-times recurrent, apparently inoperable sarcoma of the neck which had apparently entirely disappeared following an attack of facial erysipelas. Doctor Coley was very anxious to learn the end-result—no mention of it was made in the hospital history—so he patiently traced the man from one tenement house to another until he finally located him and learned that he was still in excellent health seven years after the attack of erysipelas. This observation led him in 1891 to start a series of experiments in which he attempted to produce erysipelas in patients suffering from inoperable cancer. Doctor Bull was greatly interested in the work and made it possible for him to treat 12 patients at the New York Cancer Hospital (now the Memorial Hospital) in a special pavilion erected by Mr. Archer M. Huntington. After considerable experimentation, a preparation consisting of the killed cultures of erysipelas combined with the Bacillus prodigiosus, was produced which was found to have a marked inhibitory influence upon certain types of malignant tumors, especially sarcoma. This is the preparation known as Coley's toxins, which is in use today. From the very beginning, however, Doctor Coley advocated the toxins only for inoperable sarcoma or as a prophylactic measure

after operation for bone sarcoma, and not for malignant tumors in general. In 1909, in an address before the Royal Society of Medicine, he gave a detailed description of his method of treatment and the results obtained with it; and in the discussion which followed, Sir Henry Butlin in commenting on the different kinds of so called "cancer cures" stated: "With the exception of Doctor Coley's method, scarce a single one has survived." Doctor Coley's faith in the toxins never wavered, and the large number of five year recoveries of inoperable malignant tumors that he was able to report in his address before the Royal College of Surgeons of England in October, 1935, was convincing proof that his faith had been well founded. At that meeting he was elected to honorary fellowship in the Royal College of Surgeons.

Most of Doctor Coley's work with inoperable malignant tumors was carried on at the Memorial Hospital, with which institution he was connected for more than 40 years, and on the Executive Committee of which he was still serving at the time of his death. His interest in this field was so keen and contagious that in 1902, one of his patients, Mrs. C. P. Huntington, influenced more or less by his enthusiasm, donated the sum of $100,000 for the establishment of the Collis P. Huntington Fund for Cancer Research of the Memorial Hospital—the first cancer research fund ever started in America.

It is rarely that an individual excels in more than one field; but very early in his career Doctor Coley achieved great success in operating for the radical cure of hernia. After reading his report (1897) of a series of 360 antiseptic operations for the radical cure of hernia, in which only one death had occurred and that from ether anesthesia given to a child with weak lungs, Lord Lister, at an important function in London, made the following comment: "An achievement like that is enough to cause gladness in the heart of any man who loves his fellow man. I cannot help remarking that such results could not have been obtained by the mere recognition of the truth of the importance of antiseptic principles. Such success implied that the operator was not only convinced of the truth of those principles, but also that he vigilantly maintained throughout his operations that earnest care which is necessary to prevent those principles being contravened."

Doctor Coley was one of the first in America to adopt the Bassini method of operating for the radical cure of hernia (1890) and in his recent contribution to the Bassini Bicentennial Volume compiled by the surgeons of the University of Padua, Italy, he reiterates his faith in the operation and maintains that it still remains the method of choice in most countries.

After serving as associate and then attending surgeon of the Hospital for Ruptured and Crippled, in 1924 Doctor Coley was appointed Surgeon-in-Chief, which position he held until 1931, when he became Emeritus. In 1908, he was appointed the first surgeon of the New York Central Railroad, serving as Chief Surgeon until January, 1932, when he had reached the retirement age. Another of his professional connections which was a source of great pleasure and satisfaction to him was the Mary McClellan Hospital

in Cambridge, New York, built by one of his classmates, Mr. Edwin McClellan in 1918. Doctor Coley helped to plan the hospital, to obtain an excellent resident staff, and in every way to maintain the highest possible standards for a small hospital in a rural community. As Surgeon-in-Chief, he made weekly and later monthly visits, doing much of the major surgery. He enjoyed coming in contact with the family doctor of these rural communities and was unstinting in his praise of the latter's sterling qualities.

A glance at his bibliography shows him to have been a prolific writer. His contributions deal not only with hernia and cancer, but include many essays in other surgical fields.

English literature was his greatest hobby; to him the great masterpieces of the world, apart from their solace and charm, were the master instruments of a solid education.

In 1897, at the age of 36, Doctor Coley was elected a fellow of the American Surgical Association—the youngest surgeon ever to have been elected. In 1902, he became a fellow of the Southern Surgical and Gynecological Association. He was a charter member of the American College of Surgeons; in addition he held membership in the New York Surgical, the New York Pathological, the Radiological, and the Harvard Medical societies, as well as in the American Association for Cancer Research, the American Society for the Control of Cancer, and the Association of Surgeons of Great Britain and Ireland (Honorary).

The same rich fulness, which crowned his professional life was apparent in his personal life as well. In 1891, he was married to Miss Alice Lancaster, of Newton, Mass. She and their two children, Dr. Bradley L. Coley and Mrs. William Boone Nauts, survive him. One son, Malcolm, died in his sixth year.

We who were associated with Doctor Coley, and who recognized the sterling qualities of his rare personality, realized that there was one quality which surpassed all others, even his boundless optimism, and that was his ever-readiness—in fact, eagerness—to lend a helping hand. None appealed to him in vain. He loved his fellowmen.

In closing, I would repeat the lines uttered at the time he received his degree of Honorary Master of Arts from Harvard University in 1911 (one year after his Alma Mater had conferred the same degree):

"William Bradley Coley, surgeon, medical discoverer, and director of medical research; who learned to cure by surgery ills that had foiled its art, and without surgery others beyond its reach."

Carl G. Burdick

JOHN FOX CONNORS

1873–1935

JOHN FOX CONNORS was born in Tipperary, Ireland, on October 26, 1873, and died in New York January 5, 1935. He received the degree of

JOHN FOX CONNORS, M.D.

B.A. from St. John's College in Brooklyn in 1892, and the degree of M.D. from the University of New York in 1895. He served on the house staff of Charity (now Metropolitan) Hospital and from 1898 to the day of his death

was engaged in the practice of medicine in New York. In 1892, he received the Master's degree, and in 1929 the honorary degree of L.L.D. from his Alma Mater. In 1903 he was appointed visiting surgeon to Harlem Hospital and in 1922, when elected a fellow of the American Surgical Association, he became its surgical director. He was also consulting surgeon to the Stamford Hospital and to the hospital in Waterbury, Conn.

Both as resident, and later as visiting surgeon, Doctor Connors enjoyed a varied and active service, in which major traumatic surgery largely preponderated. This was especially the case in Harlem Hospital which receives a majority of its patients from the adjacent thickly populated Negro quarter, with whom the sudden flashing of the knife or razor, and the use of the pistol is proverbial. This experience was reflected in his contributions to surgery. To minimize the danger of uncontrollable hemorrhage in stab wounds of the chest he advocated prompt and wide exploration with the ligation of a possibly divided internal mammary or intercostal artery, and in the abdomen he reported a large number of lesions of the spleen and other important abdominal viscera. His contributions to the treatment of empyema were also noteworthy.

"Jack" Connors was intensely proud of his nationality. During the customary annual dinner some years ago of the American Surgical Association, the singing of the "Wearing of the Green" by one of the invited guests was preceded by a request for any Irishman to make his presence known. Immediately "Jack" Connors stood up, and as quickly resumed his seat amidst the applause of his colleagues. He possessed the virtues of those who hail from the "Emerald Isle" with none of their vices. He was a politician, not so much to advance his own interests as those of his friends. Once a friend, always a friend; most cordial and anxious to be of service to his fellow man, and never animated by a spirit of revenge or enmity.

His surgical skill was of a high order, both in actual therapy as well as in surgical technic and judgment. His loss is keenly felt by many friends, who will always affectionately remember him. What greater tribute could one wish to leave as a heritage?

Ellsworth Eliot, Jr.

LEONARD FREEMAN

1860–1935

On December 16, 1860, there was born to Dr. Zoeth Freeman and Ellen Ricker Freeman at Pine Grove, near Cincinnati, Ohio, a son Leonard. Dr.

Leonard Freeman, M.D.

Zoeth Freeman practiced medicine in Cincinnati for years, and later became the personal physician to President U. S. Grant. During the years of his

Presidency they, with their son Leonard, lived in Washington. These years of residency in Washington during the stirring post-war period were a stimulating influence to the youthful Leonard.

Leonard Freeman received his primary education from tutors and in private schools. In 1882, he was graduated from the University of Cincinnati with the degree of B.S. In his youth he became interested in archeology, an interest he retained throughout his life. He studied ornithology with Charles Drury, a prominent naturalist of Cincinnati, whom he accompanied on several field trips, the most extended being a summer spent in the Everglades, Florida. His early studies made him a keen observer, a faculty he further developed and which later became valuable in the practice of his profession. An example of the correlation of his ornithologic and surgical observations is found in his comparison of the normal stomach of a granivorous bird with the stomach in congenital pyloric stenosis, an original, plausible explanation of the baffling etiology of this condition. (Colorado Medicine, vol. 20, p. 78, March, 1913.)

Fond of the out of doors, a lover of nature imbued with the spirit of adventure, he was prompted to take many trips into the wilds of Michigan and Canada. Over fifty years ago with two classmates, Otis Cameron and Fred Sampson, he made a canoe trip from Cincinnati up the Miami and Erie Canal to Lake Erie, thence through the Great Lakes to Duluth, Minnesota, truly an undertaking worthy of a "Voyageur."

He studied medicine at the Medical College of Ohio where he took his medical degree in 1885. He served as intern in the Cincinnati Hospital. The next three years were spent abroad at the University of Göttingen. He studied pathology under Virchow, bacteriology with Koch, and then pursued postgraduate clinical work in Vienna and Berlin.

Returning to Cincinnati he taught pathology and bacteriology in the Ohio Medical College and served as pathologist and bacteriologist to the Cincinnati Hospital from 1889 to 1891. During these years he was associated with Dr. Phineas S. Connor, then one of the ranking surgeons of the United States.

In 1891, his health broke and he went to Colorado. On regaining his strength he took a sea voyage on a sailing vessel to Honolulu. While in the Hawaiian Islands he spent some time in the Leper Colony at Molokai. On his return to Cincinnati, in 1894, he married Miss Amanda Frank. In 1895, he went to Denver, Colorado, to live. They had three sons: Frank, the eldest, an engineer living in Denver; Paul, who died in 1917; and Leonard, Junior, a surgeon who was associated with his father. His wife died in 1904. In 1906, he married Miss Jean Wright of Denver.

In 1897, Doctor Freeman became a member of the faculty of the Denver and Gross Medical College and continued to serve on the faculty when that school merged with the University of Colorado. For years he was professor of surgery and Head of the Department of Surgery of the University of Colorado Schools of Medicine.

On December 27, 1935, Dr. Leonard Freeman died of coronary thrombosis at his home in Denver, Colorado. A long, active, and productive professional career was closed at the age of seventy-five, after an illness of but

a few days. From the day of his graduation from the Medical College up to the day of the onset of his brief but fatal illness he assiduously studied, and impressively taught, practical surgery.

Dr. Freeman was a man of vigorous and powerful physique, possessed of the spirit and determination of the true pioneer. Cast in a big mold, bigness was expressed in his every thought and deed. Unostentatious, guileless, devoid of pettiness and self-aggrandizement, he could not comprehend the absence of these qualities in others, therefore he was frequently imposed upon by those less sincere and less frank.

A man of strong convictions, he not only conceded but defended the right of others to enjoy the same privilege. His brusque manner was but a superficial affectation; a defense reaction to mask a sensitive nature and a big tender heart. Only those who really knew Doctor Freeman could realize how easily he was moved and how deeply he could feel. He had schooled himself in the control of his soul-stirring emotions. He was the personification of honesty, the soul of honor and justice, aggressive and courageous, a staunch defender of the weak and a champion of the righteous.

Doctor Freeman was an ardent student of primitive as well as contemporaneous surgery, both foreign and American. Blessed with an analytical, receptive mind and an unfailing memory, with his splendid training in pathology and bacteriology, it was but natural that he became and was for many years one of America's outstanding resourceful surgeons.

He was a keen observer, a clear and logical thinker and a forceful, terse speaker. What he said or whatever he did was based upon knowledge and personal experience. In his studies, writing, teaching, consultation, operations and discussions he demonstrated a rare faculty of grasping essentials. He faced facts. He was authority.

Doctor Freeman was a world traveler. On numerous journeys through Europe, Central and South America, on voyages to Japan, China, the Philippine and South Sea Islands he never failed to study the hospitals and surgeons of these often remote countries. He availed himself of every opportunity to delve into primitive and aboriginal surgery. These studies resulted in several important papers on "Primitive and Aboriginal Surgery." His contributions to surgical literature were numerous, of wide range and valuable.

He was ex-president, a consistent attendant, and a stimulating scientific contributor of the Denver Clinical and Pathological Society, the Medical Society of the City and County of Denver, the Colorado State Medical Society and the Western Surgical Association. He was an enthusiastic member of the American Surgical Association. He was also a member of the International Surgical Association and the American College of Surgeons.

The high regard with which Doctor Freeman is held throughout the West is evidence of his excellent surgery and his stimulating influence on the vast number of his former students and the younger members of the profession.

Doctor Freeman is dead, but the memory of so great a surgeon, so inspiring a teacher, so true a man, and such a loyal friend can not die.

CASPER F. HEGNER.

MALCOLM LaSALLE HARRIS

1862–1936

Malcolm LaSalle Harris died in the Milwaukee Sanitarium at Wauwautosa, Wisconsin, on March 22, 1936, following a long illness resulting from a cerebral hemorrhage and secondary complications. Doctor Harris was

Malcolm LaSalle Harris, M.D.

born June 27, 1862, in Rock Island, Illinois, the son of Samuel G. and Frances Green Harris. He attended the public schools in Iowa and in 1882, when he was 20 years old, was graduated from Rush Medical College. After

serving his internship in the Cook County Hospital, he began practice in Chicago in 1884, where he continued to reside up to the time of his death, teaching in the County Hospital and also serving as Professor of Surgery in the Chicago Polyclinic.

Following reorganization of the American Medical Association in 1901, Doctor Harris became a member of the House of Delegates and served the American Medical Association continuously in various capacities up to the time of his incapacity due to illness. He had been successively a member of the House of Delegates, a member of the Board of Trustees, and Chairman of the Judicial Council. He was elected President of the American Medical Association in 1928, serving during 1929 and 1930. He had previously, 1898–1899, been secretary of the Section on Surgery and Anatomy, at a time when Dr. William J. Mayo was chairman of the Section. He was a member of the Board of Trustees of the American Medical Association from 1903 to 1918, and a member of the Judicial Council from 1918 to 1928.

As a surgeon, Doctor Harris had been honored with membership in the International Surgical Association, the American Surgical Association, the Western Surgical Association, and the American Association for Clinical Surgery. He served as President of the Chicago Medical Society, the Chicago Surgical Society, the Chicago Pathological Society, and the Western Surgical Association. At the Henrotin Hospital he served as Secretary of the Board continuously from 1889 to 1935 and was instrumental in the financing and construction of the new Henrotin Hospital. His contributions to medical literature include not only the translation and editing of Braun's "Local Anesthesia," and contributions to the Oxford, Keen's and Bryant's "Systems of Surgery," but also many periodical contributions not only in surgery but also in the field of medical education and more recently of medical economics.

His knowledge of the literature of surgery was catholic and his discussion of surgical questions was precise and illuminating and devoid of loose thinking and generalization. During his many years of service at the Cook County Hospital his work was eagerly attended by the younger members of the staff who recognized in him a surgeon of unusual skill and seasoned judgment. Doctor Harris was elected to membership in the American Surgical Association in 1900 and continued in active membership until the year preceding his illness. He will be greatly missed by his colleagues and many friends, for he was a man of wide acquaintanceship and recognized everywhere as a capable leader in medical, civic and financial affairs.

VERNON C. DAVID, M.D.

CARL ARTHUR HEDBLOM

1879–1934

Carl A. Hedblom was born in Boone, Iowa, on March 5, 1879. He died suddenly on June 6, 1934, at Toronto, Canada, while attending a meeting of the American Surgical Association. His death was caused by coronary thrombosis and occurred after an illness of 48 hours. The news of his sudden

Carl Arthur Hedblom, M.D.

death came as a great shock to his many colleagues and friends throughout this country and Europe.

Doctor Hedblom was educated at Colorado College, from which he received a degree of B.A. in 1907. In the following year he received a degree of M.A. and in 1921 an honorary D.Sc. from the same college. He was graduated from the Harvard Medical School in 1911 and served his internship and surgical residency under Dr. Maurice Richardson at the Mas-

sachusetts General Hospital from 1911 to 1913. In 1913 he went to Shanghai, where he was professor of surgery at the Harvard Medical School in China until 1916.

He entered The Mayo Foundation as a fellow in surgery on October 1, 1916. He was head of the section on general and thoracic surgery at The Mayo Clinic from January 1, 1919, to September 15, 1924. He received his degree of Ph.D. in surgery from the University of Minnesota in 1920. He was associate professor of surgery of The Mayo Foundation, Graduate School, University of Minnesota, in charge of the division of thoracic surgery during 1923 and 1924. He was professor of surgery at the University of Wisconsin from September, 1924, to February, 1926, when he accepted the position of professor of surgery at the University of Illinois, which position he occupied at the time of his death.

He was under appointment as Major, Medical Corps, United States Army, and was ordered to report for duty at the time of the armistice which concluded the World War.

Doctor Hedblom was a member of the Society of Clinical Surgery, of the American Surgical Association, of the Western Surgical Association, and of the American Medical Association. He was a member and past president of the American Association for Thoracic Surgery, a fellow of the American College of Surgeons and a member of the Alumni Association of The Mayo Foundation, of Sigma Xi, and of Phi Beta Kappa.

Few surgeons have had a broader training or more extensive experience in surgery than had Doctor Hedblom. In the last 15 years of his life his interest was confined chiefly to thoracic surgery. He entered this field during its early development in this country and obtained an international reputation for his work. It was fortunate for this branch of surgery that a man of his judgment, courage and conservatism became interested in it at an early date, for he was a tireless worker as well as an extensive writer and did much to stabilize the fundamental principles underlying surgery of the thorax. One of his most important contributions to the principles of thoracic surgery was to emphasize the importance, when possible, of dividing extensive operative procedures into stages. Those who were fortunate enough to know Doctor Hedblom will always remember his strong character, his frank friendliness, his cheerful smile and hearty laugh. These characteristics, combined with his inherent honesty and sincerity of purpose, earned for him the greatest admiration and respect of his colleagues and the confidence of his patients. His memory will always be a source of inspiration to progress and achievement.

In 1913, shortly before Doctor Hedblom completed his service in the Massachusetts General Hospital, he married Eleanor Pease, who with three sons and a daughter survive him.

Stuart W. Harrington, M.D.

AUGUST FREDERICK JONAS

1858–1934

August Frederick Jonas was born in Arlington, Wisconsin, in 1858. He died from cerebral hemorrhage at his home in Omaha, Nebraska, on November 12, 1934. Seven years previously he had suffered from a small cere-

August Frederick Jonas, M.D.

bral hemorrhage which left an aphasia which caused his retirement from active work.

He was graduated from the Bennett Medical College in Chicago in 1877 and took up general practice in Wisconsin. In 1882, he went abroad and studied in Vienna, Berlin and Paris for a short time and then entered the Ludwig Maximilian University in Munich as a student and was graduated in 1884.

Returning to his home state of Wisconsin he again carried on a general practice until 1887 when he moved to Omaha and became connected with the Union Pacific Railroad, eventually becoming Chief Surgeon of the road and holding this position for 29 years, until his retirement because of ill health. He served also as division surgeon for the Chicago and Northwestern Railroad and as assistant surgeon for the Chicago, St. Paul, Minneapolis and Omaha Railroad.

Being interested in teaching, Doctor Jonas joined the faculty of the Omaha Medical College as Professor of Clinical Surgery in 1892. In 1899, he added to his duties as a teacher the responsibilities of the deanship of the college and remained in that position until 1902 when the college was merged with the Medical Department of the University of Nebraska. He remained as Professor of Surgery and Chairman of the Department until his retirement in 1929, when he was given the appointment of Professor Emeritus. In 1927, he received the degree of Doctor of Laws from the State University.

Doctor Jonas was a member of many medical societies and was a conscientious attendant at meetings. He contributed to the societies by reading papers, entering freely into discussions, and serving in an official capacity when called upon. He was elected a Fellow of the American Surgical Association in 1901 and served as vice-president in 1906. He was a member of the Société Internationale de Chirurgie. At one time he was vice-president of the American Medical Association. He served for five years on the Board of Governors of the American College of Surgeons. He was president of the Western Surgical Association in 1901 and at various times served as president of the Missouri Valley Medical Society, the Nebraska State Medical Society and the Omaha-Douglas County Medical Society.

Doctor Jonas was essentially a clinical surgeon. Coming to Omaha in the early years of the city he identified himself with the surgical progress of the Middle West and gave of himself unsparingly to the efforts of the community to found hospitals and establish a medical college which would be a credit to scientific medicine.

Doctor Jonas' contributions to medical programs were concise, simply stated and written in a way to be understood readily. As he was an accomplished linguist, speaking both French and German almost as well as he did English, his acquaintance with foreign surgical literature was considerable and he added to his understanding of foreign methods by frequent trips abroad and personal contact with leaders in surgery.

As a teacher Doctor Jonas was highly respected by his students. His command of words was such that he was an adept in explaining the points that he wished to emphasize in such a way as to make the subject easily under-

standable. Consequently his clinics and ward rounds were well attended and popular with his students.

During the World War Doctor Jonas was commissioned Captain in the Medical Reserve Corps. He was active in organizing an ambulance company connected with Base Hospital No. 49 and acted as medical adviser to the Governor of Nebraska on military matters.

Doctor Jonas was a great believer in travel as a means of education. He regularly took a vacation each year and traveled to many parts of the world. On these trips he gathered material which appealed to his many interests. His collections, though not large, were brought together with discrimination, and are of great interest. They include paintings, Chinese jades, objects from the tombs of old Egypt, Alaskan Indian material and a large collection of geologic specimens from many countries. The acquisition of these collections enlarged his interests and made him a man of broad culture well grounded in many fields outside of his own science.

It is given to but few men to leave as great an impress on the community in which they lived as did Doctor Jonas. He came to a young and growing part of our country and by constant striving and continued hard work became an integral part of it and did much toward raising the standard of surgery during its period of rapid growth.

Doctor Jonas is survived by his widow, two sons and a daughter. His eldest son, August Frederick Jonas, Jr., is at present one of the residents in the Department of Surgery at Johns Hopkins University.

Alfred J. Brown, M.D.
Omaha, Nebraska.

EDWARD STARR JUDD

1878–1935

Since this Association last met surgery has sustained a heavy loss—Dr. Edward Starr Judd passed away on November 29, 1935, after a short illness. An intimate friend of all of us, his loss is an extremely personal one.

Edward Starr Judd, M.D.

We all recognized his loyalty to friends and the principles for which he stood. He inspired confidence and imparted wisdom in discussions and in those matters in which he was called to make a decision. Shy in manner, but forceful in action, he was often the arbiter in discussion. His surgical

ability was universally recognized. Possessed of rare technical ability, an exceptionally keen diagnostic sense and unusual surgical judgment, which in most instances seems to be a gift rather than an acquisition, he had the mental equipment and manual dexterity of a Master Surgeon.

Anything that might be said on this occasion would be inadequate, knowing him as we did, to emphasize the greatness of the man. Record should be made of his accomplishments for the oncoming surgeons, so that they may know of the course he pursued in the development of those talents which were given him and of his contributions to organized medicine and surgery.

He was born in Rochester, Minnesota, on July 11, 1878, and was graduated from the University of Minnesota School of Medicine in 1902. After serving an internship in St. Mary's Hospital at Rochester, he became an assistant of Dr. C. H. Mayo's in 1903.

He successively passed through the grades of the hospital and medical school staffs, and at the time of his death was surgeon to St. Mary's Hospital, professor of surgery in the graduate school of the University of Minnesota, and head of a section in the division of surgery at the Mayo Clinic.

Early in his professional career he became interested in organized medicine. The Minnesota State Medical Society recognized his worth and made him its president. He served as Secretary of the Section of Surgery of the American Medical Association during the period from 1913 to 1916, and chairman of the section in 1918. He was a member of the Council on Scientific Assembly from its beginning in 1915 to 1927. His sound advice was often sought in discussions concerning the organization and policies of this Council, which is an outstanding example of the value of demonstrations in the dissemination of the knowledge of newer things and the necessity of postgraduate teaching. The American Medical Association, realizing the outstanding service that he had rendered medicine, both in a professional and organizing way, made him President-Elect in 1930. In this office he served with high distinction, ever mindful of the needs of the members of this organization and the objectives which they should hope to obtain if they were to render the highest type of medical service.

Constant demands were made upon him to appear before medical societies. Facts were presented so clearly by him in such a simple but striking way that he became a postgraduate teacher of great renown. He stood on solid ground surgically and was never greatly moved by surgical fads. Having the gift of critical ability, he dealt with fundamentals. Listeners knew that he spoke with the authority of experience and strove to sit at his feet.

He was a member of the American Surgical Association, the American College of Surgeons, the Minnesota Academy of Medicine, the Minnesota Pathological Society, the Western, Southern and Interurban Surgical Associations, the American Society of Clinical Surgery and the Southern Minnesota Medical Association, and an honorary and corresponding member of several foreign surgical societies.

During the World War he was active as a teacher in the School for developing surgeons which was established at the Mayo Clinic, and from time to time since the war he has taken part in work designed to give those continuing in the Medical Corps of the United States Army a more intimate knowledge of diagnostic and operative procedures.

In 1921, Doctor Judd was made a member of the Editorial Board of the Archives of Surgery, and has served in a like capacity upon a number of other publications. His was a stabilizing influence, which few possessed. Although simple and retiring, he possessed great wisdom. His contributions to surgery were numerous, and those who practise surgery in the future will come to realize that he was responsible for many diagnostic methods and technical procedures, especially those relating to the biliary and gastro-intestinal tract. His memory will be kept alive by the works he has produced.

Dean Lewis, M.D.

JAMES TATE MASON

1882–1936

By the death of James Tate Mason on June 20, 1936, the surgical profession lost one of its most energetic and lovable characters. Doctor Mason was born at Lahore, Orange County, Virginia, on May 20, 1882. His grand-

McBride Studio

James Tate Mason, M.D.

father, Captain Claiborne Rice Mason was one of Stonewall Jackson's engineers during the Civil War and later was instrumental in building the Virginia Midland Railroad; in addition, he constructed the greater part of the Chesapeake and Ohio line. Doctor Mason's father, Dr. Claiborne Rice Mason, also served under Stonewall Jackson until wounded and taken to Elmira where he was kept a prisoner of war until the surrender at Appomattox. Doctor

Mason's mother, Mary Moore Woolfolk, was a native of Orange County, Virginia.

Doctor Mason attended the Locustdale Military Academy until 1901, when he entered the University of Virginia Medical School, graduating in 1905. During his school and college years, he forecast his later popularity by being greatly in demand among his mates; active in athletics, a natural leader, he was a favorite with both schoolfellows and instructors. During the summer vacations he served as an assistant to Colonel James A. Frazier, owner and manager of the Rockbridge Alum Springs, thus cheerfully helping to make his way through school. Following his graduation in 1905, he passed the examination of the Virginia State Medical Board in Richmond and began a Junior Internship in the Philadelphia Polyclinic, now known as the Post-Graduate School of the University of Pennsylvania. A residency at the Municipal Hospital of Philadelphia followed.

In the spring of 1907, as ship's surgeon, Doctor Mason came around the Horn and arrived in Seattle in the summer of the same year. Shortly thereafter, he became surgeon for the Pacific Coast Coal Company with mines at Franklin and Black Diamond, and in Franklin he did general practice for two years, making—as he did throughout his life—many staunch friends. In 1909 he moved to Seattle and shortly thereafter became physician to the county jail and, two years later, Coroner of King County. In 1914 he was appointed superintendent and Chief Surgeon of the King County Hospital which position he held until 1920.

On January 3, 1912, he married Laura DeWolfe Whittlesey of Seattle. Mrs. Mason and three children, James Tate Mason, Mary Virginia Mason and Frederick DeWolfe Mason, survive him.

In 1917, Doctor Mason and his colleagues began the association which culminated in the Mason Clinic, and within two years they organized and built the Virginia Mason Hospital of which he was Chief Surgeon and President at the time of his death.

From 1923 to 1926 he served as Secretary of the Section on Surgery, General and Abdominal, of the American Medical Association, and was Chairman of that section for one year. Between 1928 and 1934 he was a member of the House of Delegates, and during the year prior to his death, he was active in fulfilling the obligations of President-elect of the American Medical Association. His death followed some five weeks after the unusual honor of his installation in absentia to the office of President.

In addition to his local societies, Doctor Mason was a regular attendant at the meetings of the Western Surgical, Southern Surgical and Pacific Coast Surgical Associations. He was a Fellow of the American College of Surgeons and was elected to membership in the American Surgical Association in 1930. He served as President of the American Association for the Study of Goiter and of the Pacific Coast Surgical Association.

His death occurred at the Virginia Mason Hospital in Seattle on June 20, 1936, the result of endocarditis with multiple emboli.

His affiliations bear witness to his lively interest in and enthusiasm for all things surgical, and to his executive ability as well. They can give no picture, however, of his love for people which was perhaps his outstanding characteristic. His companionability was felt by all, even though their contacts with him were brief. His adaptability, keen memory for faces and names, genial manner, ready speech and an inexhaustible fund of Southern stories, all combined to endear him to a huge circle of friends. Able surgeon, executive and administrator, friend to many, his death leaves a gap in the surgical profession.

Howard C. Naffziger, M.D.

FRANCIS STUART MATHEWS

1869–1936

In the death of Francis Stuart Mathews on February 17, 1936, the surgical profession of New York City lost one of its outstanding members,

Francis Stuart Mathews, M.D.

a man combining ripe experience and rare skill with a lovable, inspiring character.

He was born in Washington, Pa., on November 14, 1869. His father, William Johnston Mathews, a storekeeper, came from a family of Scotch-Irish Presbyterians who had been settled in Pennsylvania for several generations. His mother, Frances Sage Pelletreau Mathews, was of Huguenot stock, whose ancestors had emigrated to Southampton, Long Island, following the massacre of St. Bartholomew. A forbear in whom Doctor Mathews took particular interest was his great-grandfather, Elias Pelletreau, a colonial silversmith of Southampton, examples of whose work are on exhibition in the Metropolitan Museum of Art in New York City.

When Doctor Mathews was five years old his father died and his mother with her four children went to live with an uncle in the little pioneer community of Prairie du Sac, Wisconsin. There he attended the district school in winter and worked on his uncle's farm in summer. During this period he developed an interest in birds which remained a lifelong source of study and pleasure.

At the age of 14 he was sent back to Pennsylvania to attend preparatory school and college. After taking his bachelor's degree from Washington and Jefferson in 1890, he entered the College of Physicians and Surgeons, New York City, and was graduated in medicine in 1893. The next three years he spent at Roosevelt Hospital, the first two on the surgical service of Dr. Charles McBurney and the third as resident on the gynecologic service.

With the background of a devout home, a youth in which there was a full share of plain living and hard work, and an excellent medical education, he was ready to start practice.

For 12 years he worked in the gynecologic clinic at Roosevelt Hospital. He also assisted the late Dr. Edwin B. Cragin in private practice. When Doctor Cragin ultimately offered to make him an associate at the Sloane Hospital for Women, it was a difficult decision for Doctor Mathews, as its acceptance meant limiting himself to this specialty. He chose to remain in general surgery, but the experience gained in gynecology stood him in excellent stead throughout his subsequent career.

At the outset of his practice he was appointed assistant surgeon at St. Mary's Free Hospital for Children. Within a short time he became pathologist as well, a post he held for eight years. The training gained in this position as well as at the Woman's Hospital, where he worked in pathology for two years, gave him a grounding which few surgeons acquire.

He continued on the staff at St. Mary's, becoming attending surgeon, a position he held until the hospital closed its doors. After he had been in practice five or six years he was made an assistant surgeon at General Memorial Hospital and some years later at St. Francis Hospital in the Bronx.

These latter two positions he relinquished when, in 1913, he was appointed to the surgical staff at St. Luke's Hospital. This was a fortunate move both for St. Luke's and himself. His growing reputation added to the prestige

of the hospital while he in turn found a congenial field and fuller scope for the development of his career. His private practice, already well established, increased steadily with a large following among both profession and laity.

The intense activity of these fruitful years was interrupted by beginning attacks of angina pectoris eight years before his death, when he was 58 years old. From this time on he gave up routine ward duty, but in spite of several periods of absence from work on account of ill health he retained a large practice and was in increasing demand as a consultant.

Doctor Mathews maintained a connection with his alma mater, the College of Physicians and Surgeons, first as instructor in gynecology, and later as clinical professor of surgery.

His achievements were recognized by membership in the New York Surgical Society, which he served as president, the American College of Surgeons, the Interurban Surgical Society and the American Surgical Association.

As a surgeon, Doctor Mathews had an eminently rational and scientific point of view and was never led away by fads of the moment. His judgment was sound and his conclusions, based on broad knowledge and large experience, often seemed intuitive to his assistants. He was never one to operate first and diagnose afterward, but on the other hand made up his mind promptly and boldly, where boldness was called for. He was a deft, practically ambidextrous operator, never seeming to hurry but finishing in a minimum of time. His incisions were moderate in size, the work to be done was accomplished as simply as possible without fussiness or hesitation and, as a result of gentle and expeditious handling, the patient usually made a notably uneventful convalescence.

Doctor Mathews was a frequent, although not voluminous contributor to surgical literature. His papers, covering a variety of subjects, are full of sound observations and comment and well repay rereading. Among the earlier ones is that describing his method of finger enucleation of the tonsil, developed at St. Mary's Hospital for Children at a time when tonsillectomies were frequently incompletely done. Among his later papers are two studies from his experience in the surgery of breast cancer which, thanks to the painstaking and almost complete follow up which he was able to make, as well as the uniform method of treatment carried out throughout the extensive series, are most satisfactory and authoritative contributions to the subject.

His interest in historic medicine was evidenced by a delightful paper on John of Arderne, and in the month before his death he gave no less than three talks on cultural medicine before different groups.

People trusted Doctor Mathews because of his integrity. They knew that his judgments were based on the merits of a case without consideration of self interest. They loved him because of his humanity, kindliness and devotion to their interests. The physicians who depended on his help in

their surgical problems, and the patients who benefited from his ministrations, were alike enthusiastic in his praise.

His professional success depended not one iota on pushing aggressiveness. On the contrary, he was modest and retiring and the hospital appointments and honors which came to him were offered in recognition of his abilities and not sought out. He completely lacked the egotism and acquisitiveness which characterize many successful men, but strove rather to share opportunities and push the interests of those associated with him.

This thoughtfulness for others and desire to help them was one of his outstanding characteristics. He was always ready to assist a younger colleague in a case, if by this support the patient could be satisfied, rather than have him fall into his own hands. On his hospital service he saw to it that his assistants had as many and as interesting cases as it was possible to pass on to them.

One of Doctor Mathews' most delightful qualities was his keen sense of humor with which his conversation sparkled. He was a man of broad culture and could quote the Bible and Shakespeare in an inimitable way.

Although he was devoted to his profession and found his chief joy in it to the day of his death, he had other interests. Reference has already been made to his knowledge of birds. This was enhanced by trips to the West Indies where the bird life attracted him as well as the history of the Islands. He took a keen interest in the American Museum of Natural History where he was named research associate in experimental biology. He was a deacon in the Presbyterian Church.

In 1923, his alma mater, Washington and Jefferson, conferred on him the degree of Doctor of Laws.

His wife, the former Miss Julia E. King, of Columbus, Ohio, survives him as do their three children. He was fortunate in having his older son, Dr. Frank P. Mathews, associated with him in practice during his last years.

On February 17, 1936, after his usual hospital visit in the morning, he was examining a patient in the office when stricken with an attack of coronary thrombosis. He was relieved of pain by sedatives and was apparently resting comfortably that evening, when, in the midst of a conversation with his physician he suddenly died. It was an ending which one might envy, yet with his splendid faculties unimpaired, how much there is to regret that he could not have been spared a longer time.

Morris K. Smith, M.D.

LORD MOYNIHAN

1865–1936

Berkeley George Andrew Moynihan, Honorary Fellow of the American Surgical Association, was born in Malta on October 2, 1865. His father

Berkeley George Andrew Moynihan, M.D.

was Captain Moynihan, V. C., of the 8th Foot (The King's Regiment). Moynihan went to the Royal Naval School and received his medical education

at Leeds and Berlin. He qualified in 1887 with the M.B., B.S. degree, London, and received the diplomas of the Royal Colleges. He took the F.R.C.S. in 1890, and in the examination for the M.S.Lond, in 1893, was awarded a gold medal. After residence in the General Infirmary at Leeds as house-surgeon and resident surgical officer he was appointed to its honorary surgical staff in 1896, and was also demonstrator in the anatomy department of the Yorkshire College Medical School. Later he became Professor of Surgery in the University of Leeds. Honors were showered upon him from all parts of the world. His own city of Leeds conferred upon him the honorary freedom of the city; he was made Vice-Chancellor of the University of Leeds on the occasion of its jubilee in 1924; and he was elected a vice-president of the British Medical Association in its centenary year, 1932. He was knighted in 1912. He was made a C.B. in 1917, a K.C.M.G. in 1918, and a baronet in 1922. In March, 1929, he was created Baron Moynihan of Leeds.

He was President of the Association of Surgeons of Great Britain and Ireland, 1922-1923, President of the Section of Surgery at the annual meetings of the British Medical Association in 1925, 1930, and 1932, and President of the Royal College of Surgeons from 1926 to 1932. He died on September 7, 1936, at his home, Carr Manor, Leeds, after a brief illness.

Moynihan was unique because of his contributions to surgery, his surgical craftsmanship, his writing, his oratory, and his leadership. In each of these he was so outstanding that any one quality, as he possessed it, would have made his name famous.

His earliest major contribution was during his association with the late W. Mayo Robson when together they wrote a book on "Diseases of the Stomach" in 1901. The more notable contributions during his active surgical career were "The Pathology of the Living," "Abdominal Operations," "Gallstones and Their Surgical Treatment," and "Duodenal Ulcer." One of his most charming essays, "Truants in Medicine," appeared in recent months and illustrated his interest in the history of medicine.

As a surgical craftsman he was superb. Many Fellows of the American Surgical Association have thrilled at the perfection of his technic and the mastery he displayed in every operation he undertook. The "Lancet" in commenting on Moynihan as a craftsman has said: "It would be difficult to overestimate the influence Moynihan had on English surgical craftsmanship. It was not that he originated new details in technique, though he was insistent on the great importance of attention to detail; or that he invented surgical instruments, though there are many with which his name is associated. His effect was above all a psychological one. He brought about a general alteration in the intellectual attitude to operations. Perhaps it is more true to say that his forceful personality drove out into the open the faith of the many conscientious surgeons whose natural reserve prevented open confession. He regarded every operation almost as a religious rite; as he ex-

pressed it, 'a sacrament.' He felt with great intensity the magnitude of the surrender of a man's whole future, and indeed very often of his life, to the judgment and manual skill of a perhaps hitherto unknown surgeon. That was the gospel he preached and on these foundations was raised the magnificent edifice of his surgical technique. By his personality not only did he infuse a like spirit into the surgeons and accessory staff with whom he was associated, but he was able to communicate it to visiting and other surgeons who carried it to their own centres of work. In countless operating theatres procedure has been based on the example at Leeds."

His dominant personality was as evident in his writing as in the operating room. He believed that the English language afforded greater opportunity for clear exposition than any other language, and he so carefully cultivated the art of writing that the pen in his hands became an instrument to give such beauty of expression and clarity of thought as to substantiate this belief.

As an orator Moynihan had an ease of delivery which could only come from ceaseless application and cultivation of a natural endowment and which gave to his addresses a charm unequalled. This alone captured his audiences, and with his ability to discover unusual and remarkable phrases and his extraordinary knowledge of his subject, he became the great orator of his day in medicine.

His leadership was shown in the founding of "The British Journal of Surgery," in his efforts to develop a postgraduate school in London, and in the organization of the Moynihan Chirurgical Club. He made many visits to America, and on one of these visits he, with Sir William Taylor and Mr. Albert Carless, presented to the American College of Surgeons at its meeting in Montreal a ceremonial mace upon which appears the following dedicatory inscription: "From the Consulting Surgeons of the British Armies to the American College of Surgeons, in memory of mutual work and good fellowship in the Great War, 1914-1918." In addition to being an Honorary Fellow of the American College of Surgeons, he served as Consulting Editor for "Surgery, Gynecology and Obstetrics."

It is futile to attempt a short sketch of the life of a man of such attainments, for these will be the evidence of the influence of Moynihan on surgery and surgeons of this generation.

Donald C. Balfour, M.D.

WILLIAM JOHNSON TAYLOR

1861–1936

WILLIAM JOHNSON TAYLOR, a Fellow of The American Surgical Association since April, 1887, died at his home in Philadelphia January 22, 1936.

WILLIAM JOHNSON TAYLOR, M.D.

He was born in Worcester County, Md., October 13, 1861. He was graduated from the Medical School of the University of Pennsylvania in 1882

and then served a two year internship at the Pennsylvania Hospital. A few years later he became Assistant to W. W. Keen in his private work and at the Orthopedic Hospital and Infirmary for Nervous Diseases. It was at this hospital that S. Weir Mitchell and W. W. Keen developed a large neurosurgical clinic and Taylor was Keen's right-hand man from that time until the latter's retirement. He thus assisted Keen in all his early brain operations. Later he succeeded Keen as surgeon to the hospital. Doctor Taylor served the Orthopedic Hospital continuously and conscientiously for 50 years, a service which was gratefully acknowledged in 1935 by both the Board of Managers and the Staff of the hospital. In addition to his service at the Orthopedic Hospital he was for many years surgeon to St. Mary's and St. Agnes' Hospitals.

Doctor Taylor was what might well be styled a sane surgeon. He was well trained in the art and was a capable and conscientious operator. He was held in very high regard not only by his friends and colleagues but also by the general profession, largely because of his fine character and his well known stand on matters relating to professional and educational standards.

His greatest professional interest was the College of Physicians. He served this institution for a long period of years on various committees and conspicuously on the Building and Finance Committees. He was made President of the College in 1909 and occupied the chair for three years. He was very active in the Philadelphia Academy of Surgery, being Secretary for many years and President in 1908–1909.

An interest outside of medicine to which he gave a great deal of his time was the Philadelphia Library, an old and honorable institution founded by Benjamin Franklin. Doctor Taylor was President of the Board for two years.

When the United States entered the World War, Doctor Taylor was 56 years of age. He accepted the rank of First Lieutenant and went to France with the Pennsylvania Hospital Unit, Base Hospital No. 10, which on arrival in France was assigned to the British service and given General Hospital No. 16 at La Tréport. He was with this Base Hospital throughout his service and was advanced to Captain September 10, 1917, and to Major January 9, 1918. On November 11, 1918, he was made Lieutenant Colonel. His war service was characterized by the same earnest enthusiasm that was conspicuous in his professional life both before and after the War.

Sympathy, kindliness and consideration of others were outstanding characteristics of William Johnson Taylor. In 1891, Doctor Taylor married Miss Emily Buckley Newbold and she with their four children survive him.

John H. Gibbon, M.D.

JONATHAN MAYHEW WAINWRIGHT

1874–1934

By the death of Jonathan Mayhew Wainwright on August 3, 1934, the American Surgical Association lost one of its most valued members. He was Vice President of the Association in 1930.

Jonathan Mayhew Wainwright, M.D.

Doctor Wainwright was born February 20, 1874, in Hartford, Connecticut; the son of William Augustus Muhlenberg Wainwright, M.D., whose father was Bishop Jonathan Mayhew Wainwright. His mother was Helena Barker Talcott. He came from old New England stock and was descended from nine colonial governors. He was educated at Hartford High School, and received his B.A. degree at Trinity College, Hartford;

and his M.D. degree at the College of Physicians and Surgeons, New York, in 1899. Trinity College later conferred upon him an honorary M.A. degree. He served as intern at St. Luke's Hospital in New York from 1899 to 1901 and was married immediately upon leaving the hospital to Jessie Bell Hart of Englewood, N. J. While an intern in the hospital he showed deep interest in his medical work and serious study of the problems thereof. In the few hours for recreation which an intern might find in the evening, he occupied himself in the laboratory, cutting and studying pathologic slides which even then formed the nucleus and ground work for the study which he carried on through the rest of his medical career.

Immediately upon finishing his internship, although a young man, he became Surgeon-in-Chief at the Moses Taylor Hospital in Scranton, Pa., which position he occupied up to the time of his death. He served during the Spanish-American War as Adjutant in the First Connecticut Regiment. In the World War, he served overseas successively as Major, Lieutenant Colonel and Colonel in the Medical Corps, and received a citation from General Pershing.

Doctor Wainwright was not only an earnest student but a prolific contributor to general surgical literature, as well as many public health problems. He was always interested in cancer research, particularly in cancer of the breast, and carried on a correspondence and made a study of not only his own cases, but also of material supplied to him by his surgical colleagues in other cities. His slides of complete cross sections of carcinomatous breasts were beautifully prepared and most carefully studied by him. He was President of the American Society for Control of Cancer in 1930, also Chairman of the Pennsylvania State Cancer Commission, the first in the field of state work. He was director of the Cancer Research Clinic, of Scranton, Pa.

In 1930, he made a four-months' trip through Uganda, Kenya and Tanganyika for the purpose of cancer research among the native African tribes. In addition, as evidence of his broad interest in other problems of public health, he organized, in 1902, the West Mountain Sanatorium, in Scranton, for the treatment of tuberculosis, and in 1907 he made a study of, and reported on, the Scranton typhoid epidemic which had occurred in the previous year.

While Doctor Wainwright's chief interest was always some phase of the cancer problem, and although during his later years most of his surgical contributions pertained to this subject, particularly breast cancer, a list of his surgical contributions covers almost every phase of general surgery.

His scientific mind, the concentration of purpose with which he would pursue a problem, his devotion and interest in his professional work, made him an outstanding surgeon, and one of the most valuable men to both the profession and public of his adopted state of Pennsylvania. His many friends both in the American Surgical Association and in the profession at

large, as well as of the public generally, have lost one who will be hard to replace.

It is indeed an ironical and sad commentary on the progress of the knowledge of the disease to which he had devoted so much of his life's study, that this same disease should have progressed to such a degree in his own case, that the condition was inoperable before a diagnosis could be made.

JOHN DOUGLAS, M.D.

INDEX

A

Acute perforation of gastrojejunal ulcer, 388
Address of the President: the making of a surgeon, 1
Adson, Alfred, discussion on essential hypertension, 311
Alexander, John, discussion on thoracoplasty, 97
some advances in the technic of thoracoplasty, 72

B

Balfour, Donald C., memorial of Berkeley George Andrew Moynihan, 441
Beer, Edwin, delayed wound healing following nephrectomy for tuberculosis, 275
discussion on transurethral resection, 336
Benign cicatricial strictures of the bile ducts, 195
Benson, Clifford D., a ten year study of empyema in children, 106
Bergh, George S., cholecystoduodenostomy combined with pyloric exclusion, 229
Blalock, Alfred, discussion on chronic constrictive pericarditis, 55
discussion on regional enteritis, 142
Bloodgood, Joseph Colt, memoir, 413
Boland, Frank K., traumatic surgery of the lungs and pleura, 99
Brown, Alfred J., memorial of August Frederick Jonas, 428
Burdick, Carl G., memorial of William Bradley Coley, 415

C

Cabot, Hugh, discussion on nephrectomy for tuberculosis, 278
discussion on transurethral resection, 340
Carter, B. Noland, the late results of Thoracoplasty in the treatment of pulmonary tuberculosis, 79
Cholecystoduodenostomy combined with pyloric exclusion, 229
Churchill, Edward D., discussion on chronic constrictive pericarditis, 56
pericardial resection in chronic constrictive pericarditis, 43
Coley, William Bradley, memoir, 415
Coller, Frederick A., discussion on rectal cancer, carcinoma of the rectum, 177
the one stage procedure of the treatment of carcinoma of the rectum, 163
Connors, John Fox, memoir, 419
Crile, George W., discussion on essential hypertension, 308
Curtis, George M., splenectomy for hemoclastic crises, 370

D

David, Vernon C., discussion on peritonitis, 260
discussion on rectovaginal cloaca, 185
memorial of Malcolm LaSalle Harris, 424
Delayed wound healing following nephrectomy for tuberculosis, 275
Demonstration of hormones in tumors, the, 314

Doan, Charles A., splenectomy for hemoclastic crises, 370
Douglas, John, discussion on strictures of the bile ducts, 225
memorial of Jonathan Mayhew Wainwright, 446

E

Edelman, Leo, delayed wound healing following nephrectomy for tuberculosis, 275
Eliot, Jr., Ellsworth, benign cicatricial strictures of the bile ducts, 195
discussion on strictures of the bile ducts, 226
memorial of John Fox Connors, 419
Estes, Jr., William L., discussion on gastrojejunal ulcer, 126
Experimental lymphedema of the intestinal tract and its relation to regional cicatrizing enteritis, 128

F

Finney, John M. T., memorial of Joseph Colt Bloodgood, 413
Freeman, Leonard, memoir, 421

G

Gastric acidity following operations for gastric and duodenal ulcer, 112
Geschickter, Charles F., the demonstration of hormones in tumors, 314
Gibbon, John H., memorial of William Johnson Taylor, 444
Giertz, Knut H., twenty-five years' experience in the treatment of peritonitis, 239
Graham, Evarts A., discussion on tumors of the chest wall, thoracoplasty, bronchial fistulae, 94
report of the committee to study further problems of postgraduate surgical education in general and the qualifications for specialization in general surgery in particular, 12
Graham, Henry F., the influence of heredity in cancer, 383
Graham, Roscoe R., discussion on postoperative insulin crisis, 193
discussion on strictures of the bile ducts, 222
Guerry, Le Grand, discussion on postoperative insulin crisis, 194
operative insulin crisis in resection of the pancreas, 189

H

Harrington, Stuart W., discussion on tumors of the chest wall, thoracoplasty, bronchial fistulae, 92
memorial of Carl Arthur Hedblom, 426
Harris, Malcolm LaSalle, memoir, 424
Hedblom, Carl Arthur, memoir, 426
Hegner, Casper F., discussion on tumors of the chest wall, thoracoplasty, bronchial fistulae, 91
memorial of Leonard Freeman, 421
Hener, George J., the surgical treatment of essential hypertension, 298
Homans, John, discussion on regional enteritis, 141
Horsley, J. Shelton, discussion on rectal cancer, carcinoma of the rectum, 175
discussion on strictures of the bile ducts, 224

I

Imperforate anus with rectovaginal cloaca, 178
Influence of heredity in cancer, the, 383

Ivy, Andrew C., cholecystoduodenostomy combined with pyloric exclusion, 229
discussion on cholecystoduodenostomy, 238

J

Jonas, August Frederick, memoir, 428
Jones, Daniel Fiske, discussion on rectal cancer, carcinoma of the rectum, 172
Jones, T. Banford, obstructions about the mesentery in infants, 342
Judd, Edward Starr, memoir, 431

K

Kretschmer, Herman L., discussion on transurethral resection, 341
transurethral resection, 325

L

Lahey, Frank H., discussion on gastrojejunal ulcer, 127
discussion on hyperthyroidism, 41
discussion on rectal cancer, carcinoma of the rectum, 174
discussion on strictures of the bile ducts, 218
Late results of thoracoplasty in the treatment of pulmonary tuberculosis, the, 79
Lehman, Edwin P., studies in brain injury; increased cerebrospinal fluid pressure from blood in the cerebrospinal fluid, 19
Lewis, Dean, memorial of Edward Starr Judd, 431
the demonstration of hormones in tumors, 314
Lilienthal, Howard, discussion on tumors of the chest wall, thoracoplasty, bronchial fistulae, 89
Lower, William E., discussion on rectovaginal cloaca, 185
discussion on transurethral resection, 339
Lund, Charles Carroll, discussion on hormones in tumors, 323

M

Making of a surgeon, the; address of the president, 1
Mason, James Tate, memoir, 434
Matas, Rudolph, discussion on peritonitis, 260
Mathews, Francis Stuart, memoir, 437
McCutcheon, George T., operative insulin crisis in resection of the pancreas, 189
McKenna, Hugh, the reduction of the increasing mortality and morbidity in acute appendicitis, 144
Morton, John J., obstructions about the mesentery in infants, 342
Moynihan, Berkeley George Andrew, memoir, 441

N

Naffziger, Howard C., memorial of James Tate Mason, 434

O

Obstructions about the mesentery in infants, 342
Ochsner, Alton, discussion on peritonitis, 258
the prevention of peritoneal adhesions by papain, 263

One stage procedure of the treatment of carcinoma of the rectum, the, 163
Operative insulin crisis in resection of the pancreas, 189

P

Parker, William H., studies in brain injury; increased cerebrospinal fluid pressure from blood in the cerebrospinal fluid, 19
Penberthy, Grover C., a ten year study of empyema in children, 106
discussion on hyperthyroidism, 42
reactions following operations for hyperthyroidism, 34
Pericardial resection in chronic constricture pericarditis, 43
Phemister, Dallas H., discussion on essential hypertension, 312
Pickhardt, Otto C., discussion on rectovaginal cloaca, 186
Pool, Eugene H., address of the president: the making of a surgeon, 1
discussion on bronchial fistulae, 97
Prevention of peritoneal adhesions by papain, the, 263

R

Rankin, Fred W., resection of the rectum and rectosigmoid by single or graded procedures, 155
Ransom, Henry K., the one stage procedure of the treatment of carcinoma of the rectum, 163
Reactions following operations for hyperthyroidism, 34
Reduction of the increasing mortality and morbidity in acute appendicitis, the, 144
Reichert, Frederick L., discussion on regional enteritis, 143
experimental lymphedema of the intestinal tract and its relation to regional cicatrizing enteritis, 128
Reid, Mont Rogers, discussion on rectal cancer, carcinoma of the rectum, 175
Report of the committee to study further problems of postgraduate surgical education in general and the qualifications for specialization in general surgery in particular, 12
Resection of the rectum and rectosigmoid by single or graded procedures, 155
Rôle of surgery in the treatment of actinomycosis, the, 279

S

Sandblom, Philip, cholecystoduodenostomy combined with pyloric exclusion, 229
Schwyzer, Arnold, discussion on tumors of the chest wall, thoracoplasty, bronchial fistulae, 95
Scrimger, Francis A. C., a technic for the management of gastrojejunal ulcers with or without gastrocolic or jejunocolic fistula, 121
discussion on gastrojejunal ulcer, 127
Shenstone, Norman S., discussion on bronchial fistulae, 98
the use of intercostal muscle in the closure of bronchial fistulae, 87
Smith, Morris K., memorial of Francis Stuart Mathews, 437
Some advances in the technic of thoracoplasty, 72
Speed, Kellogg, discussion on tumors of the chest wall, 98
tumors of the chest wall, 57
Splenectomy for hemoclastic crises, 370
Stone, Harvey B., discussion on rectal cancer, carcinoma of the rectum, 176
discussion of rectovaginal cloaca, 188
imperforate anus with rectovaginal cloaca, 178
Storck, Ambrose, the prevention of peritoneal adhesions by papain, 263

Studies in brain injury; increased cerebrospinal fluid pressure from blood in the cerebrospinal fluid, 19
Surgical treatment of essential hypertension, the, 298

T

Taylor, William Johnson, memoir, 444
Technic for the management of gastrojejunal ulcers with or without gastrocolic or jejunocolic fistula, a, 121
Ten year study of empyema in children, a, 106
Thompson, Harold Lincoln, acute perforation of gastrojejunal ulcer, 388
Tinker, Martin Buel, discussion on tumors of the chest wall, thoracoplasty, bronchial fistulae, 91
Toland, Clarence G., acute perforation of gastrojejunal ulcer, 388
Torek, Franz, discussion on peritonitis, 257
discussion on rectovaginal cloaca, 185
Transurethral resection, 325
Traumatic surgery of the lungs and pleura, 99
Tumors of the chest wall, 57
Twenty-five years' experience in the treatment of peritonitis, 239

U

Use of intercostal muscle in the closure of bronchial fistulae, the, 87

W

Wainwright, Jonathan Mayhew, memoir, 446
Walters, Waltman, discussion on strictures of the bile ducts, 220
gastric acidity following operations for gastric and duodenal ulcer, 112
Wangensteen, Owen H., discussion on peritonitis, 259
discussion on tumors of the chest wall, thoracoplasty, bronchial fistulae, 93
the rôle of surgery in the treatment of actinomycosis, 279
Whipple, Allen O., discussion on postoperative insulin crisis, 193
discussion on strictures of the bile ducts, 219
Wiseman, Bruce K., splenectomy for hemoclastic crises, 370